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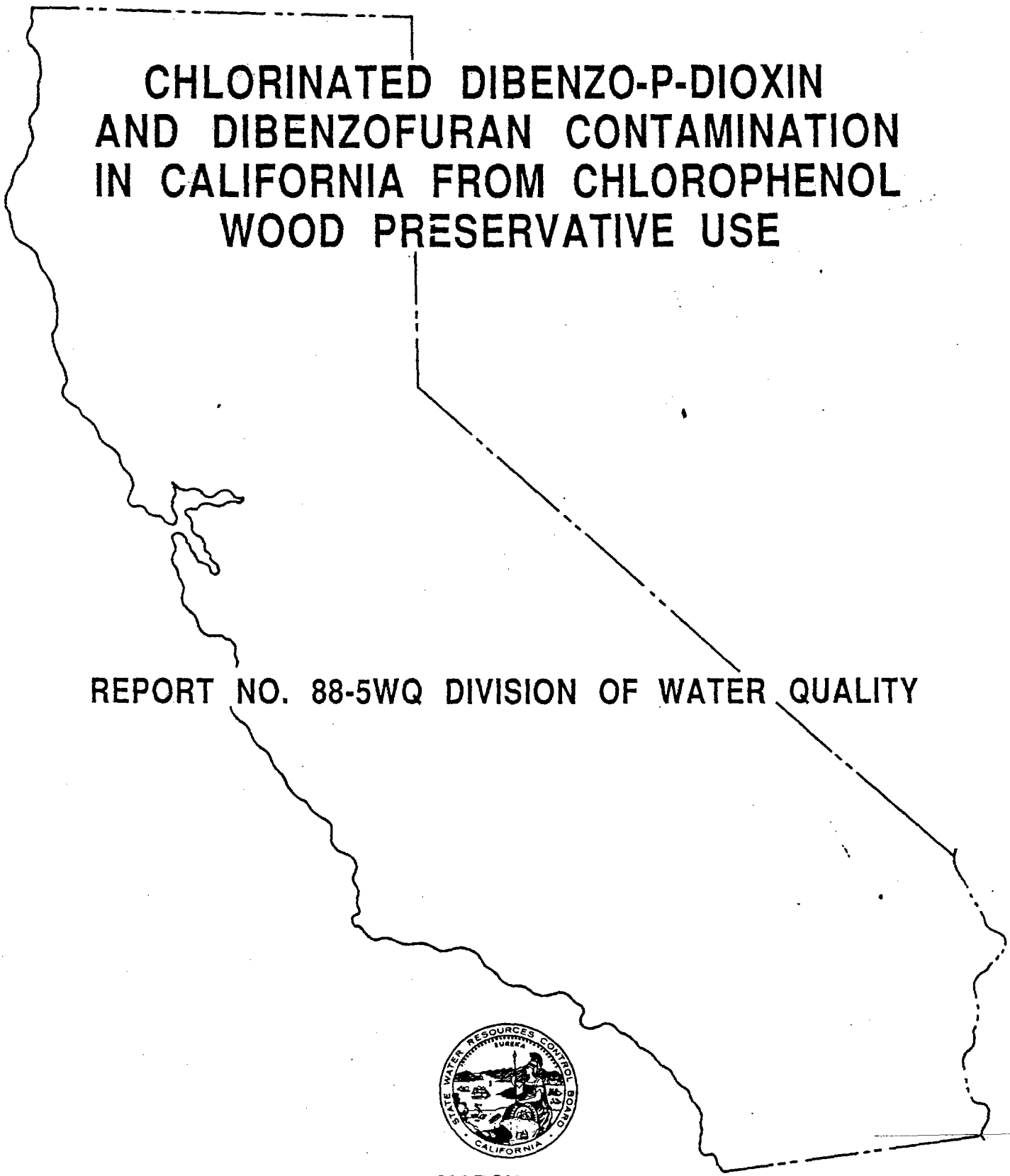
CHLORINATED DIBENZO-P-DIOXIN AND DIBENZOFURAN CONTAMINATION IN CALIFORNIA FROM CHLOROPHENOL WOOD PRESERVATIVE USE



REPORT NO. 88-5WQ DIVISION OF WATER QUALITY

MARCH 1988

STATE WATER RESOURCES CONTROL BOARD

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CHLORINATED DIBENZO-P-DIOXIN AND DIBENZOFURAN CONTAMINATION IN CALIFORNIA FROM CHLOROPHENOL WOOD PRESERVATIVE USE

REPORT NO. 88-5WQ DIVISION OF WATER QUALITY

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STATE WATER RESOURCES CONTROL BOARD

PREFACE

This report is one in a series of reports issued by the State Water Resources Control Board on industrial and agricultural chemicals. These reports deal with priority chemicals of concern to water quality and the protection of beneficial uses of water in California. In February 1982, the State Board initiated an Industrial Chemicals program based on the premise that the production and use of chemicals should not occur at the expense of water quality protection.

Chemicals are of inestimable value to society, and most are considered relatively safe under normal conditions of use. There are some chemicals whose environmental and health effects have been proven harmful. The possibility that toxic chemicals in the environment can cause cancer in humans and severely impair the health of wildlife has led to increased action by government to foster the safe use and disposal of these chemicals.

The chronic effects of persistent chemicals (e.g., impaired growth and reproduction) may be more devastating in the long run than immediately apparent effects, such as fish kills. Preventative measures are invariably less costly to society than corrective actions required after toxic chemical pollution has occurred.

Some current chemical use and disposal practices may have an adverse impact on water quality. These activities can usually be modified to minimize adverse environmental effects. Where existing or potential water quality problems have been identified, the State Board will recommend appropriate measures to correct or prevent such adverse impacts.

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LIST OF ABBREVIATIONS

Chemicals

CDD	Chlorinated dibenzo-p-dioxin
MonoCDD	Monochlorodibenzo-p-dioxin
DiCDD	Dichlorodibenzo-p-dioxin
TriCDD	Trichlorodibenzo-p-dioxin
TetraCDD	Tetrachlorodibenzo-p-dioxin
PentaCDD	Pentachlorodibenzo-p-dioxin
HexaCDD	Hexachlorodibenzo-p-dioxin
HeptaCDD	Heptachlorodibenzo-p-dioxin
OctaCDD	Octachlorodibenzo-p-dioxin
CDF	Chlorinated dibenzofuran
MonoCDF	Monochlorodibenzofuran
DiCDF	Dichlorodibenzofuran
TriCDF	Trichlorodibenzofuran
TetraCDF	Tetrachlorodibenzofuran
PentaCDF	Pentachlorodibenzofuran
HexaCDF	Hexachlorodibenzofuran
HeptaCDF	Heptachlorodibenzofuran
OctaCDF	Octachlorodibenzofuran
PCP	Pentachlorophenol
TetraCP	Tetrachlorophenol
TriCP	Trichlorophenol
NaPCP	Sodium pentachlorophenate
K-tetraCP	Potassium tetrachlorophenate
2,4-D	2,4-Dichlorophenoxyacetic acid
2,4,5-T	2,4,5-Trichlorophenoxyacetic acid
BAP	Benzo(a)pyrene
CCA	Chromated copper arsenate
DMBA	7,12-Dimethylbenz(a)anthracene
3MC	3-Methylcholanthrene
PCB(s)	Polychlorinated biphenyl(s)
PCDE	Polychlorinated diphenyl ether
TPA	12-O-Tetradecanoylphorbol-13-acetate

Terms

AAL	Applied Action Level
ADI	Acceptable daily intake
AHH	Aryl hydrocarbon hydroxylase
AWQC	Ambient water quality criteria
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act (Superfund)
CSF	Confidential statements of formula
CWA	Clean Water Act
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
GC	Gas chromatography
GC-MS	Gas chromatography-Mass Spectroscopy
HPLC	High performance liquid chromatography
LOEL	Lowest observed effect level
MFO	Mixed function oxidase
MS	Mass spectroscopy

NOAEL	No observed adverse effect level
NOEL	No observed effect level
NSPS	New source performance standards
PSES	Pretreatment standards for existing sources
RCRA	Resource Conservation and Recovery Act
SNARL	Suggested no adverse response level
STLC	Soluble threshold limit concentration
TEF	Toxic equivalency factor
TTLC	Total threshold limit concentration

Units

ppm	parts per million
ppb	parts per billion
ppt	parts per trillion
ppq	parts per quadrillion
g/l	gram per liter
mg/l	milligram (10^{-3} grams) per liter (equal to ppm)
ug/l	microgram (10^{-6} grams) per liter (equal to ppb)
ng/l	nanogram (10^{-9} grams) per liter (equal to ppt)
pg/ml	picogram (10^{-12} grams) per milliliter (equal to ppt)
ug/m^3	microgram (10^{-6} grams) per cubic meter
pg/m^3	picogram (10^{-12} grams) per cubic meter
g/kg	gram per kilogram (equal to parts per thousand)
mg/kg	milligram per kilogram (equal to ppm)
ug/kg	microgram per kilogram (equal to ppb)
pg/g	picogram (10^{-12} grams) per gram (equal to ppt)

Government Agencies, Groups, and Private Industries

CAC	California Administrative Code
CARB	Calif. Air Resources Board
CAG	Carcinogen Assessment Group (U.S. EPA)
CDC	Center for Disease Control
CDWG	Chlorinated Dioxin Work Group (U.S. EPA)
CDHS	Calif. Department of Health Services
CVRWQCB	Central Valley Regional Water Quality Control Board
DWR	Calif. Department of Water Resources
U.S. EPA	United States Environmental Protection Agency
FDA	United States Food and Drug Administration
IARC	International Agency for Research of Cancer
NAS	National Academy of Science
NCI	National Cancer Institute
NIOSH	National Institute for Occupation Safety and Health
NRCC	National Research Council of Canada
NTP	National Toxicology Program
SCE	Southern California Edison
USFWS	United States Fish and Wildlife Service
WHO	World Health Organization

Symbols

>	greater than
≥	greater than or equal to
<	less than
≤	less than or equal to

GLOSSARY

Most terms defined in the glossary are specific to usage in this report.

acute toxicity--involving a stimulus severe enough to rapidly induce an adverse response; in toxicity tests, a response observed in 96 hours or less is typically considered acute. Acute toxicity is most often reported in terms of lethality (e.g. LC50), but various other adverse effects may be measured (e.g. EC50).

adenoma--a benign neoplasm of glandular epithelial tissue.

adipose tissue--tissue in which fat is stored.

adsorb--the assimilation of gas, vapor, or dissolved matter onto a solid surface.

Ah receptor(aryl hydrocarbon receptor)--a soluble protein in the cell cytoplasm capable of binding an aromatic hydrocarbon molecule and inducing synthesis of gene products of the Ah locus.

Ah locus--gene complex responsible for the synthesis of aryl hydrocarbon hydroxylase (AHH) and several other enzymes.

aliphatic--a term applied to the "open chain" or fatty series of hydrocarbons; non-ring organic compounds.

alopecia--baldness;absence of hair from skin areas where it normally is present.

antigen--a substance capable of inducing the formation of antibodies in the blood.

Aroclor--trade name for a group of polychlorinated biphenyls; e.g. Aroclor 1242 indicates 12 carbon atoms and 42% chlorine by weight.

benign--not malignant; a benign tumor will not metastasize, a malignant tumor will.

bile--a fluid secreted by the liver that aids in digestion.

bilirubin--a reddish, yellow pigment in bile derived from hemoglobin during red blood cell (RBC) destruction.

bioaccumulation--uptake, concentration, and retention of substances by an organism from its surrounding medium and from food.

bioassay--a test used to evaluate the relative potency of a substance by comparing its effect on a living organism with the effect of a standard preparation on the same type of organism.

bioavailability--the degree to which a drug or other substance is available to the target tissue after administration or to organisms in the environment.

bioconcentration--uptake and concentration of a substance from the surrounding medium through gill membranes or epithelial tissue.

bioconcentraion factor(BCF)--in standard tests, the ratio at equilibrium of the concentraion of a substance in the tissue of a test organism to its concentration in the surrounding medium. BCF is substance- and species-specific.

biotransformation--the series of chemical alterations of a compound which occur within an organism by enzymatic activity (sometimes causing the resulting compound to be more toxic, sometimes less.)

capillary column--a long open tube of small diameter having the inside wall coated with a thin film of stationary phase; used in gas chromatography for the separation of closely spaced peaks.

carcinogen--a cancer-producing substance.

carcinoma--a malignant growth derived from epithelial tissue and tending to infiltrate the surrounding tissue.

catabolism--any destructive process by which complex compounds are broken down into more simple substances.

caudal fins--tail fins of fish and aquatic mammals.

cell-mediated immunity--specific acquired immunity in which the role of small lymphocytes of thymic origin is predominant; it is responsible for resistance to infectious diseases caused by certain bacteria and viruses.

chloracne--a skin lesion resembling acne caused by exposure to chlorinated compounds.

choline kinase--an enzyme that transfers a high energy group, such as phosphate, to choline, an amino alcohol and a member of the vitamin B complex.

chromatid--one of a pair of "sister" chromatids, identical connected nucleoprotein strands, products of replication of the parent chromosome, that are joined at the centromere and separate during cell division, each becoming a chromosome of one of the two daughter cells.

chronic toxicity--toxic effects from a prolonged exposure of an organism to sublethal amounts of a toxic substance, often one-tenth of the life span or more.

clean-up--laboratory purification of a sample before further analysis.

coelute--simultaneous desorption of two or more analytes from an analytical column such that they are not separated at the detector.

comedo(comedones-plural)--a plug in an excretory duct of the skin, containing microorganisms and keratin; also called a blackhead.

congener--refers to any one particular compound of the same chemical family; e.g. there are 75 congeners of chlorinated dibenzo-p-dioxin.

conjugate--a biochemical reaction product combining a foreign or natural compound or its metabolite with an endogenous carbohydrate, protein, or sulfur derivative.

cytochromes--any of a class of hemoproteins whose principal biological fuction is electron transport.

cytoplasm--the protoplasm (viscous, collodial semifluid) of a cell exclusive of the nucleus.

depurate--to be removed or reduced in concentration in a medium over time, as the result of a metabolic or physical process.

desorption--removal of a substance from an adsorbed state by physical or chemical process.

EC 50 (effective concentration)--the concentration of a substance in food and water at which 50% of the organisms treated exhibit the measured effect.

edema--the presence of abnormally large amounts of fluid in the intercellular tissue spaces of the body.

embryotoxicity--stillbirth or in utero death during the embryonic stage before the placenta is completely formed, which in humans is approximately the first 8 weeks after conception.

endoplasmic reticulum--an ultra microscopic organelle of nearly all cells of higher plants and animals consisting of a more or less continuous system of membrane-bound cavities that ramify throughout the cytoplasm of the cell.

enzyme induction--increased activity of the enzyme systems upon exposure to chemicals.

epidemiological--relation of the various factors determining the frequency and distribution of diseases within a given population.

epoxide--cyclic ethers; an atom of oxygen bound to two separate carbons which are linked, forming a three-membered ring.

eutrophication--the natural process of aging of bodies of water resulting in an increase in mineral and organic nutrients such as nitrogen and phosphorus, and reduced levels of dissolved oxygen. Eutrophic lakes may be characterized by algal or bacterial blooms and diminished fish life.

extraction--separation and isolation of analytes from a sample matrix, usually through the use of solvents.

femto-(f)--indicates one-quadrillionth (10^{-15}); for example, there are one million billion (10^{15}) femtograms in a gram, or one billion billion (10^{18}) femtograms in a kilogram.

fetotoxicity--stillbirth or in-utero death during the fetal (post-embryonic) stage.

fibrosarcoma-a malignant neoplasm derived from fibrous connective tissue.

fractionation--a step in sample preparation for analysis which separates the analytes contained in a sample into multiple fractions which have similar physical and chemical properties.

frameshift mutation--addition or deletion of base pairs on the DNA molecule. If the number of base pair changes is not a multiple of three, the amino acid sequence of the proteins coded after the mutation is drastically changed.

gavage--feeding through a tube passed through the mouth into the stomach.

gene--the biological unit of heredity. A functional segment of DNA on a chromosome which codes and regulates production of one or more specific proteins.

genotoxicity--the effect of a substance that interacts with and alters DNA or RNA; when the DNA or RNA is replicated the alteration is carried on.

glucuronide--any compound containing glucuronic acid which is a tetrahydroxy-aldehyde acid.

glutathion-s-transferase--a family of enzymes that catalyzes glutathion conjugation.

gravid--pregnant;containing developing young.

half-life--the time in which the concentration of a substance will be reduced to one-half of its initial value through degradation or elimination from the medium.

hematologic--pertaining to blood.

hemoprotein--a conjugated protein containing heme as the prosthetic group, e.g. hemoglobin.

hepatic--pertaining to the liver.

humoral immunity--aquired immunity in which the role of circulating antibodies (immunoglobulins) is predominant.

hyperpigmentation--abnormally increased pigmentation.

hyperplasia--an abnormal increase in the size of a tissue or organ due to an increase in the number of normal cells.

ID_{50}--(immunological dose) a dose producing 50 percent suppression of the immune system.

immunoglobulin--an antibody synthesized by special lymphocytes (plasma cells) in response to the introduction of an antigen.

immunosuppression--the artificial inhibition of the immune system.

immunotoxic--quality of a substance which interferes with the immune system.

in utero--within the uterus.

in vitro--within an artificial environment, e.g. biochemical studies in laboratory glassware.

in vivo--within a living body.

initiation--interaction of a carcinogen with a normal cell to produce a cancerous or precancerous cell.

integumentary system--an enveloping layer (as a skin or membrane) of an organism or one of it parts.

intraperitoneal--injection into the abdominal cavity that is lined by a serous sac (peritoneum).

isomer group--a group of structurally related chemicals (isomers) with the same molecular formula, e.g. there are eight isomer groups of CDDs, monochlorinated through octachlorinated.

isomers--chemical compounds that have the same molecular formula but different molecular structures or different arrangements of atoms in space; refers to substances which belong to the same homologous class; e.g. 22 isomers constitute the homologue of tetraCDD.

kinetic--refers to the processes and rates of chemical reactions.

LC_{50}--the lethal concentration (LC) of a toxicant in food or water to 50% of the exposed population.

LD_{50}--the lethal dose (LD) of a toxicant to 50% of the exposed population.

leachate--water that has percolated through soil containing soluble substances and that contains certain amounts of these substances in solution.

lignin--a polysaccharide which, in connection with cellulose, forms the cell wall of plants and wood.

lipid--fatty material characterized by the presence of fatty acids or their derivatives and by their solubility in non-polar solvents.

lipophilic--readily soluble in lipid.

liquid column chromatography--an analytical procedure where a sample or sample extract is passed through a column containing an absorbent which selectively entrains the analyte(s) of interest for later analysis.

log K_{ow}--logarithm of the partition coefficient between octanol and water for a given substance.

lymphoma--a general term applied to any neoplastic disorder of the lymphoid tissue.

mRNA(messenger ribonucleic acid)--a form of RNA in living cells that is responsible for carrying the genetic code transcribed from DNA to specialized sites within the cells for the synthesis of polypeptides.

malignant--an abnormal growth that tends to spread to other sites.

mass:charge ratio--in mass spectroscopy, the ratio of the mass of a fragment ion to its electronic charge.

mass spectrometer--an analytical instrument in which an analyte molecule is fragmented to produce a pattern of ions which is used for either identification or quantification.

maxilla--the iregularly shaped bone, composed of two maxillae joined together to form the upper jaw.

metabolite--any chemical substance produced by metabolism or by a metabolic process, e.g. breakdown products from biochemical reactions.

mexacarbamate--a carbamate pesticide.

micro-(u)--indicates one-millionth (10^{-6}); for example, there are one million (10^{6}) micrograms in a gram, or one billion (10^{9}) micrograms in a kilogram.

microbial degradation--the breakdown of compounds by microscopic organisms.

milli-(m)--indicates one-thousandth (10^{-3}); for example, there are one thousand (10^{3}) milligrams in a gram or one million (10^{6}) milligrams in a kilogram.

mixed function oxidases(MFO)--enzyme systems found predominantly in the endoplasmic reticulum of the liver which have a form of cytochrome P-450 as the terminal oxidase.

mutagen--a physical or chemical agent that alters DNA or RNA.

mutagenicity--the ability of a substance to alter DNA or RNA.

nano-(n)--indicates one-billionth (10^{-9}); for example, there are one billion (10^{9}) nanograms in a gram, or one trillion (10^{12}) nanograms in a kilogram.

necrosis--death of tissue.

neoplasm--new and abnormal growth, such as a tumor, that may be either benign or malignant.

neoplastic nodule--abnormal swelling or protuberance.

neuropathy--a general term denoting functional disturbances and/or pathological changes in the peripheral nervous system.

NOAEL--no observed adverse effect level; synonymous with NOEL.

NOEL--no observed effect level; the highest measured continuous concentration of an effluent of a toxicant that does not cause health effects or clinical signs on a test organism.

nuclear magnetic resonance--an analytical method for identification of atomic constituentsof chemicals, using knowledge of absorption of electromagnetic radiation at a precise frequency by the atomic nucleus.

oligotrophic--a body of water with a poor supply of nutrients and a low rate of formation of organic matter by photosynthesis.

opercular--pertaining to an operculum which is a lid or flap of skin covering an opening or orifice, e.g. the gill cover of fishes.

organic--denoting chemical substances containing the element carbon.

pancytopenia--deficiency of all cell elements of the blood; aplastic anemia.

parenchymal cells--cells in loose connective tissue whose function is to pack the space between organs.

partition coefficient--the ratio of a chemical distributed between two parts of a system such as octanol and water or sediment and water.

peri position--chlorines in the 1,4,6,& 9 position on a CDD or CDF molecule.

photodegradation--chemical decomposition induced by light.

photolysis--chemical reaction involving bond-cleavage produced by exposure to light or ultraviolet radiation (adjective: photolytic).

photosensitize--to make an organism or chemical sensitive to light.

pico-(p)--indicates one-trillionth (10^{-12}); for example, there are one million million (10^{12}) picograms in a gram, or one quadrillion (10^{15}) picograms in a kilogram.

pleiotrophic gene--a gene that affects a number of different characteristics in a given individual.

porphyria--any of a group of disturbances of porphyrin metabolism characterized by marked increase in the formation and excretion of porphyrin precursors.

porphyrin--any one of a group of iron-free or magnesium-free cyclic tetrapyrrole derivatives which occur universally in the protoplasm.

predioxins--chlorinated 2-phenoxyphenols.

promoter--a substance which is not directly carcinogenic, but enhances effect of carcinogenic agents.

Salmonella typhimurium--a bacterial species used in mutagenicity tests.

sarcoma--a malignant neoplasm derived from connective tissue.

serum--blood plasma minus its clotting proteins.

sister chromatid exchange(SCE)--an exchange at one locus between the sister chromatids which does not result in an alteration of overall chromosome morphology.

sludge--the semiliquid precipitate of waste treatment processes, or settled residue in tanks or ponds used for chemical treatment, e.g. material found in lumber dip tanks at wood treatment facilities.

soil column--a vertical column of soil usually a core sample, which displays horizontal layers of soil material.

solubility--the amount of a substance that can be dissolved in a given amount of solvent, normally expressed as mg/l.

soxhlet extraction--a repetitive extraction and distillation procedure for extracting analytes from a solid sample matrix.

static--at rest or in equilibrium; not dynamic.

steady state--a stable equilibrium condition of a system in which change in one direction is continually balanced by change in another.

structure-activity relationship--the biochemical activity of a compound related to its structure.

subchronic toxicity--toxic effects produced by a test compound during an exposure of intermediate duration usually lasting about three months.

subclinical--without clinical manifestations; said of the early stages, or a slight degree, of a disease.

subcutaneous--beneath the skin.

substrate--a substance upon which an enzyme or catalyst acts.

T-lymphocyte--a thymus dependent lymphocyte (a mononuclear white blood cell).

technical grade chemical--a chemical that has not been purified after production and may contain many impurities.

teratogenic--producing non-lethal morphological or functional changes in the fetus.

thymic atrophy--wasting away or decrease in size of the thymus.

thymus--a bilobed organ, located in the lower neck, that plays a role in the immune mechanism of the body.

triglyceride--a compound consisting of three molecules of fatty acid esterified to a glycerol.

turbinate--shaped like a cone; a turbinate bone located in the nasal passages.

ultraviolet (UV) spectroscopy--an analytical method that utilizes the fact that the amount of ultraviolet light absorbed by an analyte is a function of its concentration in solution.

uptake--absorption and incorporation of a substance by living tissue.

vehicle--the substance in which a compound is dissolved or mixed prior to dosing an animal with that compound.

wasting--gradual loss, decay, or diminution of bulk.

EXECUTIVE SUMMARY

1. INTRODUCTION

In 1984, the State Board began a priority chemical investigation of certain chemicals used for wood preservation at California sawmills and wood treatment plants. Pentachlorophenol, one of the most widely used wood preservative fungicides, was given special attention, as it is known to contain highly toxic byproducts produced during its chemical manufacture. These contaminants include chlorinated dibenzodioxins (CDDs) and a related group of chemicals, chlorinated dibenzofurans (CDFs). Chemical identification of these substances is extremely difficult, in part because there are so many of them (75 different CDDs and 135 possible CDFs). Only 15 of these 210 compounds (6 CDDs and 9 CDFs) are considered highly toxic. The most toxic compound is commonly referred to as "dioxin" or 2,3,7,8-tetrachlorodibenzodioxin. As "dioxin" has been studied most extensively, much of what has been estimated about the other CDDs and CDFs is based on knowledge of this compound.

The CDDs and CDFs have never been intentionally manufactured. They are only produced as reference standards which are required for chemical analysis. In addition, CDDs and CDFs are known to occur as byproducts of chemical synthesis, from electrical equipment fires, and from municipal solid waste incinerators. The CDDs and CDFs have received widespread media attention because of several incidents involving human exposures. These events include the use of the herbicide Agent Orange in Vietnam, a chemical plant explosion at Seveso, Italy, CDD-contaminated oil used for dust control in Missouri, and CDF-contaminated rice oil poisoning incidents in Japan and Taiwan.

The State Board study described in this report was designed to determine which, if any, of the 15 most toxic CDDs and CDFs were present at sawmills and wood treatment plants in California. In order to perform the difficult chemical analysis, split samples were sent to three laboratories in the United States and Sweden. Several of the 15 most toxic CDDs and CDFs were detected in samples of soil, sawmill sludges and liquids, commercial pentachlorophenol formulations, and crystals formed during wood pressure treatment.

2. ENVIRONMENTAL FATE

As a group, the CDDs and CDFs share three characteristics that make them long-lived in the environment: very low water solubility, high affinity for soil and sediment and resistance to breakdown. However, as individual compounds, the CDDs and CDFs exhibit wide diversity. For example, the eight chlorine CDD is about 100,000 times less soluble than the CDDs containing four chlorine atoms. The combination of very high toxicity and very low water solubility has made the measurement and modeling of CDDs and CDFs in the environment a difficult task. However, recent work has shed some light on a number of processes that may affect the persistence of these compounds in the environment. These include the following:

a. On soil surfaces, CDDs and CDFs can be both formed and broken down by sunlight. For example, they can be formed from the joining of two pentachlorophenol molecules, while more highly chlorinated compounds can be converted to lower chlorinated ones. Under certain conditions, the lower chlorinated CDDs and CDFs that are formed from such breakdown conversions can be more toxic than more highly chlorinated parent compounds.

b. Naturally occurring micro-organisms will not significantly breakdown CDDs and CDFs.

c. Despite having low vapor pressures, CDDs and CDFs can be transported from water and soil to the air. Detection of these compounds at clean sites is therefore strongly suggestive of atmospheric deposition.

d. CDDs and CDFs can migrate to ground water if organic solvents are also present. In the absence of organic solvents, they are not expected to migrate significantly unless "channels" such as cracks in rocks are present.

e. CDDs and CDFs will bind strongly to suspended matter in water. The major "sinks" for these compounds in water are sediments, particulates, and living organisms.

f. Because of the extremely low water solubility of CDDs and CDFs, water-based leachate tests designed to simulate conditions in a municipal landfill are not likely to detect their presence.

3. AQUATIC TOXICOLOGY

In addition to toxic effects occurring at very low (parts per trillion) concentrations, the most striking aspect about the effect of "dioxin" on aquatic life is that toxic reactions are not observed until 5 to over 100 days after exposure. An amount as low as 5.6 parts per trillion has been shown to be lethal to salmon with other toxic effects observed as low as 0.1 parts per trillion. The CDDs and CDFs also are bioconcentrated to a high degree in aquatic organisms. The highest reported bioconcentration factor is approximately 9,000 for both rainbow trout and mosquito larvae. The most toxic CDDs and CDFs are also most preferentially bioconcentrated.

As this report went to press, the State Board learned of new toxicity and bioconcentration information obtained from a recent chronic study. Published in January 1988, the study examined the effects over a 56-day period of very low levels of the most toxic CDD and most toxic CDF on rainbow trout. Levels as low as 38 parts per quadrillion of the CDD had significant adverse effects on survival and growth. CDF levels as low as 0.9 parts per trillion reduced growth and 4 parts per trillion reduced survival. Bioconcentration factors by rainbow trout also were higher than previously reported: 39,000 for the CDD and 6,000 for the CDF.

4. MAMMALIAN TOXICOLOGY

Both CDDs and CDFs are absorbed and concentrated by humans and laboratory animals. The half-life of the most toxic CDD was over five years in a human volunteer, in contrast to shorter half-lives (10 to 40 days) in laboratory animals.

The most toxic CDD is also extremely variable in lethality, depending on animal species. For example, it takes approximately 5,000 times as strong a dose to kill a hamster as a guinea pig. As with aquatic animals, death in mammals is delayed after a single lethal dose, typically between 5 and 45 days. Death occurs after a period of wasting away.

In addition to lethality, these compounds also produce long term effects. Studies with laboratory animals have shown that the most toxic CDD causes reproductive (teratogenic) and fetal (fetotoxic) defects at very low exposure levels. These effects have not, however, been observed to date after accidental human exposure. Studies of the most toxic CDD and of a mixture of two other toxic CDDs have shown these compounds to be strong animal carcinogens. The U.S. Environmental Protection Agency (EPA) has rated the most

toxic CDD as the most potent animal carcinogen ever tested. However, there is little conclusive evidence from human exposure to date that this compound is linked to human cancer. A recent newspaper account in the New York Times (December 9, 1987) noted that EPA may reduce the estimate of CDD potency by a factor of 16. If this EPA rating system estimate does change, CDD will still be the most toxic carcinogen known. At the new estimate, the "safe" daily dose would be raised to 0.1 parts per quadrillion per day based on body weight (A part per quadrillion is one divided by 10^{15}).

5. CRITERIA AND STANDARDS

Criteria and standards have been developed primarily for the most toxic CDD. For example, the U. S. Food and Drug Administration in 1983 set a safe level of 25 parts per trillion in fish for human consumption as long as fish was not consumed more than twice a month. The U. S. Centers for Disease Control recommended a site specific cleanup level of 1 part per billion in soil. There is considerable debate in the scientific community over whether the 1 part per billion level for soil cleanup is too conservative (too safe) or not safe enough.

The EPA currently considers the most toxic CDD such a strong carcinogen that the one in one million risk level is set below the current chemical detection limit. This water criterion of 0.013 parts per quadrillion is based on a daily intake by a 70 kilogram man of 2 liters of water and 6.5 grams of fish or shellfish.

6. WOOD TREATMENT PRACTICES AND CALIFORNIA SITE CONTAMINATION

Pentachlorophenol and similar compounds have been used routinely for decades at sawmills and wood treatment facilities in California. Wood is typically treated by either dipping it in tanks containing the preservative solution, by spraying, or by forcing the solution under pressure into the wood. The latter method is used at wood treatment plants to provide long lasting protection. In contrast, sawmills use the dipping or spraying methods as a shorter term means to protect the surface from fungal growths that stain the wood and degrade its market value. Typically, the areas where wood is treated have been contaminated by the treatment chemical. Where pentachlorophenol has been used, the contaminants have included CDDs and CDFs. Because of their environmental persistence, these compounds may be present many years after the use of pentachlorophenol has ceased.

This report provides three examples of contamination by pentachlorophenol in California: at Visalia, Selma, and Oroville. At the Visalia site, a plume of organic solvents transported pentachlorophenol, CDDs and CDFs into both the shallow and deep aquifers. Contamination of the deeper aquifer was especially worrisome since the City of Visalia's drinking water wells were located downstream of the site. High levels of CDDs and CDFs were detected in soil samples at Selma while extensive pentachlorophenol contamination of ground water has occurred near Oroville.

7. CALIFORNIA WATER RESOURCES CONTROL BOARD STUDY

Based on a report of high levels of CDFs found in dip tanks at two Swedish sawmills, the State Board investigated wood treatment facilities in California to determine if CDDs and CDFs were also present.

When CDDs and CDFs are found, they usually occur as a complex mixture of different compounds. The degree of difficulty of chemical analysis for CDDs and CDFs depends on the type of analysis performed. The easiest method is group analysis. For example, one group of CDDs contains six chlorine atoms. There are actually ten different CDDs with six chlorines, but, by measuring only the group, the analysis is simplified.

Testing for individual CDD and CDF compounds is much more difficult. For example, of the ten different compounds in the six chlorine group of CDDs, three are highly toxic. The most accurate approach to evaluate their toxicity would be to measure the individual concentrations of these three highly toxic compounds.

The State Board study tested both the simpler group approach as well as individual compound analysis. In the group analysis phase, 13 samples -- soil (4), sludge (4), dip tank liquid (2), and commercial pentachlorophenol (3) -- were examined for presence of CDDs and CDFs. Significant concentrations of these groups of compounds were detected in all samples, with the highest concentrations found in the commercial formulations and dip tank sludges.

In the subsequent individual compound analysis phase, 12 samples from four sites (three sawmills, and one wood pressure treatment plant) were analyzed for the 15 most toxic CDDs and CDFs. Typically, at least three of the six toxic CDDs and seven of the nine CDFs were present in sawmill sludges and commercial mixtures. A noteworthy sample was obtained at a pressure treatment plant, where the

crystals or "bloom" formed on the surface of treated lumber contained five of the most toxic CDDs and eight of the most toxic CDFs. This information indicates that highly toxic CDDs and CDFs can be present, often at significant concentrations, as contaminants at sawmills and wood treatment plants.

8. HAZARD EVALUATION

The approach used in this report is based on an interim method, published in 1986 by the EPA, to evaluate the toxicity of CDD and CDF mixtures. It follows the premise that these different compounds follow similar toxicological pathways and that their toxic effects in mixtures are additive.

Each of the 15 highly toxic CDDs and CDFs has a different estimated toxicity. The EPA approach is to assign the most highly toxic CDD (the "dioxin") a toxicity value of 1.0 units, while the remaining 14 are given values ranging from 0.001 to 0.5 units, based on available toxicity information.

The "total" toxicity of a particular mixture of CDDs and CDFs is then calculated by multiplying the toxicity value of each separate CDD or CDF by its concentration in the sample. This step is performed for each of the highly toxic CDDs and CDFs and the results are added to obtain a total toxicity concentration for the mixture. Using this method, the highest relative toxicity concentration determined in a commercial pentachlorophenol formulation was 290 parts per billion. In sawmill dip tank sludge, the relative toxicity concentration ranged from 27 to 330 parts per billion. In the crystals formed after pressure treatment, the relative toxicity concentration was calculated to be 100 ppb.

Characterization of the "total" toxicity of CDDs and CDFs in mixtures by this method allows for estimation of site specific potential hazards as well as options for remedial action. The report recommends that remedial action assessment be based upon the "Decision Tree" approach developed by the California Department of Health Services. At some sites, moving the material may create more of a hazard than on-site storage of CDD and CDF containing materials isolated from humans and the environment. The latter approach may be the most effective interim measure until acceptable methods of CDD and CDF destruction become available.

RECOMMENDATIONS

1. Sawmill sludges and soils should be analyzed for the presence of CDDs and CDFs prior to disposal.

   The CDDs and CDFs previously concentrated in dip tank sludges will remain until the tanks are cleaned. Before disposal, these sludges should be analyzed for potential presence of CDDs and CDFs. If these compounds are present, sludge disposal by land or low temperature burning should be avoided. These materials should be held in interim storage until an effective means of destruction is identified and is available.

2. Wood treatment plants should improve management practices to isolate crystals of pentachlorophenol formed after treatment.

   Crystals (or "bloom") formed on lumber after pentachlorophenol pressurized treatment contain high levels of toxic CDDs and CDFs. During sampling by State Board staff, it was observed that some of this material falls to the ground during normal operating procedures. Plant operations should be improved to prevent environmental contamination by these crystals.

3. The highest priority should be given to isolating chlorinated dibenzodioxins and dibenzofurans from the environment and destroying them.

   Over 100 million dollars has been expended worldwide for research on the most toxic CDD. Nevertheless, many questions regarding toxicity and environmental fate of CDDs and CDFs still exist. Effective means to safely degrade these compounds, such as high temperature incineration or other methods, must be developed as rapidly as possible.

4. Interim on-site storage of CDD and CDF-containing materials is recommended until effective means of destruction are developed.

   Mobility and availability of CDDs and CDFs are dependent upon site specific soil types and characteristics, annual rainfall, plant and animal populations, and bioavailability. CDDs and CDFs should therefore not be placed in landfills. If, in the future, on-site land treatment is proposed, methods must be specifically designed for each site to avoid human or environmental exposure.

5. The California Site Mitigation Decision Tree Manual (Decision Tree) should be used as guidance for clean-up of CDD and CDF-contaminated sites.

   The Decision Tree process, published by the California Department of Health Services, consists of five elements: (1) preliminary site appraisal; (2) site assessment; (3) risk appraisal; (4) environmental fate and risk determination; and (5) development of site mitigation strategies and selection of remedial action.

6. Estimates of the concentrations of the most highly toxic CDDs and CDFs in contaminated materials should be made by following procedures described in this report.

   Considering the complexity and expense of analyzing for 210 individual CDDs and CDFs, analysis should be focused on the eight groups. Then the "total" toxicity of the most toxic CDD and CDF compounds in soil and dip tank samples can be estimated by using the percentage of highly toxic compounds calculated in this report. This will greatly simplify analysis for CDDs and CDFs by identifying only the four, five, six, and seven chlorine groups for each of these two compound classes.

7. Estimation of the toxicity of CDD and CDF mixtures should follow the U.S. EPA "toxicity equivalency factor" approach.

   As an interim approach to estimating the toxicity of samples containing CDD and CDF complex mixtures, the U.S. EPA has recommended a system based on multiplying the concentrations of individual highly toxic CDDs and CDFs by respective potency factors. These factors are based on both carcinogenicity and other toxicity test values of various CDDs and CDFs relative to the most toxic CDD.

8. EPA should develop a national strategy for identifying chemicals (or classes of chemicals) that may cause toxicity beyond the normal 96 hour acute test period.

   For chemicals thus identified, EPA should recommend observation periods for acute aquatic toxicity be extended from the current 96 hour standard bioassay test to at least 30 days beyond the acute test period. These recommendations follow observations of toxic effects induced by CDDs and CDFs up to one month after the initial exposure, when mortality did not occur within the standard 96 hour test period.

9. Interim advisories for highly toxic CDDs.

Advisory limits have been proposed by the U. S. Government, by other states, and by the province of Ontario, Canada, for drinking water, fish flesh, and soil cleanup. Although not the focus of this State Board report, the starred (*) levels listed below can serve as interim guidelines for California until advisories are established by the California Department of Health Services. It should be noted that some of these advisories are at or below the current practical detection limits for these compounds.

a. 2,3,7,8-tetraCDD (the most toxic CDD)

i. Drinking Water (protection of human health)

U.S. EPA (1984)	2.2×10^{-4} ppt*	(0.2 parts per quadrillion)
National Academy of Sciences (1977)	0.7 ppt	
New York State (Ground Water-drinking water supply) (1987)	3.5×10^{-2} ppt	(35 parts per quadrillion)

ii. Fish Flesh

U. S. FDA (1983)	50 ppt*
Province of Ontario (1986)	20 ppt
Michigan (1986)	10 ppt
New York (1987)	10 ppt

iii. Soil Cleanup Level

United States Centers for Disease Control (Atlanta, GA) (1984)	1 ppb	(site-specific for Times Beach, Missouri)

b. hexaCDD (six-chlorine CDD) - Drinking Water

U.S. EPA (1985)	5.5×10^{-3}* ppt	(5.5 parts per quadrillion)

TECHNICAL SUMMARY

1. INTRODUCTION

Highly toxic compounds were found in products and environmental samples at selected California sawmills and wood treatment plants. These were chlorinated dibenzodioxins and chlorinated dibenzofurans ("CDDs" and "CDFs"). These classes of compounds include 2,3,7,8-tetrachlorodibenzodioxin, which is popularly referred to as "dioxin".

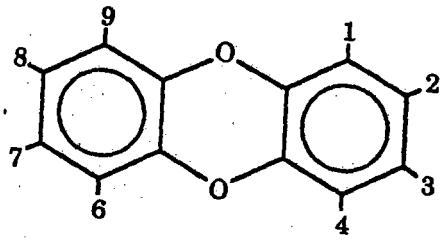
Structures of these compounds are shown in Figure 1. Dioxin has received widespread press coverage because it was a contaminant in Agent Orange, an herbicide used in Vietnam. It was detected in the streets of Times Beach, Missouri, and traced to contaminated oil used for dust control. The town was evacuated and bought out by the U. S. Government after the Centers for Disease Control determined that the 2,3,7,8-tetraCDD concentrations in soil represented an unreasonable risk to humans. Chlorinated dibenzofurans (CDFs) are contaminants in polychlorinated biphenyl (PCB) formulations. Both CDFs and PCBs contributed to significant human health problems in Japan and Taiwan. Rice oil had been accidentally contaminated with high concentrations of both compounds and was consumed by humans.

As shown in Figure 1, the chlorinated dibenzodioxin and dibenzofuran molecules each can contain from one to eight chlorine atoms. Since these can be arranged in a variety of ways, up to 75 CDDs and 135 CDFs are possible (Table 1). A mixture having both CDDs and CDFs theoretically could contain 210 individual compounds. The CDDs and CDFs having four, five, six, or seven chlorine atoms, four of which are in the 2,3,7, and 8 positions, are considered to be significantly toxic to mammals. The number of these is fifteen: six CDDs and nine CDFs (Table 2). The two eight-chlorine containing ("octa-") CDDs and CDFs also have four 2,3,7,8-substituted chlorine atoms. However, the octaCDDs and CDFs are believed to have low toxicity and in this report are not considered in the hazard evaluations of samples containing them.

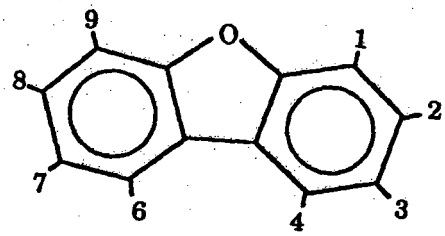
CDDs and CDFs are not produced intentionally, except as reference standards for chemical analysis. They appear, for example, as by-products of chemical synthesis, electrical equipment fires, and municipal incineration of solid wastes. They are contaminants of chlorophenol wood preservatives. In California, approximately 100 sawmills and wood treatment plants have been in operation or exist today. Almost half of these have used chlorophenol wood preservatives. These chemicals and their contaminants are present at an undefined number of sites, regardless of whether or not the plants are still operating.

FIGURE 1

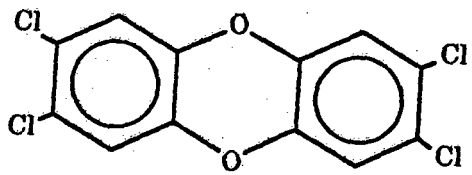
CHEMICAL STRUCTURES



Dibenzo-p-dioxin



Dibenzofuran



2,3,7,8-Tetrachlorodibenzo-p-dioxin
(2,3,7,8-TetraCDD)
("Dioxin")

NUMBERS ON STRUCTURES REFER TO LOCATION WHERE CHLORINE ATOMS CAN BE ATTACHED

TABLE 1

NUMBER OF COMPOUNDS IN CHLORINATED DIBENZODIOXIN AND DIBENZOFURAN ISOMER GROUPS

Isomer Group	Number of Compounds in Isomer Group
CDDs	
1. Monochlorodibenzodioxin (monoCDD)	2
2. Dichlorodibenzodioxin (diCDD)	10
3. Trichlorodibenzodioxin (triCDD)	14
4. Tetrachlorodibenzodioxin (tetraCDD)	22
5. Pentachlorodibenzodioxin (pentaCDD)	14
6. Hexachlorodibenzodioxin (hexaCDD)	10
7. Heptachlorodibenzodioxin (heptaCDD)	2
8. Octachlorodibenzodioxin (octaCDD)	1
TOTAL CDD COMPOUNDS	75
CDFs	
1. Monochlorodibenzofuran (monoCDF)	4
2. Dichlorodibenzofuran (diCDF)	16
3. Trichlorodibenzofuran (triCDF)	28
4. Tetrachlorodibenzofuran (tetraCDF)	38
5. Pentachlorodibenzofuran (pentaCDF)	28
6. Hexachlorodibenzofuran (hexaCDF)	16
7. Heptachlorodibenzofuran (heptaCDF)	4
8. Octachlorodibenzofuran (octaCDF)	1
TOTAL CDF COMPOUNDS	135
CDD AND CDF TOTAL	210

TABLE 2

2,3,7,8-CHLORINE SUBSTITUTED DIBENZODIOXINS AND DIBENZOFURANS

Isomer Group	Total Compounds in Isomer Group	Number of Compounds in Isomer Group with 2,3,7,8 Substitution	Specific Isomers
CDDs:			
Tetra-	22	1	2,3,7,8-tetraCDD
Penta-	14	1	1,2,3,7,8-pentaCDD
Hexa-	10	3	1,2,3,4,7,8-hexaCDD
			1,2,3,6,7,8-hexaCDD
			1,2,3,7,8,9-hexaCDD
Hept-	2	1	1,2,3,4,6,7,8-heptaCDD
Octa-	1	1	1,2,3,4,6,7,8,9-octaCDD
Total tetra through octaCDD compounds	49	7	
CDFs			
Tetra-	38	1	2,3,7,8-tetraCDF
Penta-	28	2	1,2,3,7,8-pentaCDF
			2,3,4,7,8-pentaCDF
Hexa-	16	4	1,2,3,4,7,8-hexaCDF
			1,2,3,6,7,8-hexaCDF
			1,2,3,7,8,9-hexaCDF
			2,3,4,6,7,8-hexaCDF
Hepta-	4	2	1,2,3,4,6,7,8-heptaCDF
			1,2,3,4,7,8,9-heptaCDF
Octa-	1	1	1,2,3,4,6,7,8,9-octaCDF
Total tetra through octaCDF compounds	87	10	

2,3,7,8-TetraCDD is the most potent animal carcinogen ever evaluated in the laboratory. EPA has estimated that this compound is approximately 20 and 50 times more potent than the next two highest-ranked carcinogens (a mixture of two hexaCDDs and Aflatoxin B_1, respectively). It is 50 million times more potent than trichloroethylene (TCE) or vinyl chloride. This 2,3,7,8 four chlorine-containing compound also is highly acutely toxic to certain animal species. A single feeding of one part to one billion parts body weight will kill half of a guinea pig test population.

The findings of dramatic CDD acute toxicity and carcinogenicity in animals contrasts with the lack of comparable findings in humans. Over one hundred million dollars has been spent over the last few decades studying the toxicity and fate of principally one compound, 2,3,7,8-tetraCDD. Large gaps in knowledge still exist. The most prudent approach at this time should be minimizing CDD and CDF entry into the environment. This is an alternative to continuing to spend large sums of money on research that produces as many questions as answers.

The present State Board study detected CDDs and CDFs at sawmills and wood treatment plants in soils and dip tank liquids and sludges. CDDs and CDFs were present where pentachlorophenol had been used for wood preservation. Most of the toxic CDD and CDF compounds listed in Table 2 were detected in all samples. To our knowledge, this is the first study in the United States which has identified the fate of individual 2,3,7,8-substituted CDDs and CDFs in chlorophenol wood preservatives after their use. The analytical chemistry necessary to perform such detailed trace analysis involved three laboratories in the United States and Sweden.

2. ENVIRONMENTAL FATE

The anticipated stability and distribution of CDDs and CDFs depends upon the individual compound, environmental conditions, and the nature of experiments designed to predict its environmental fate. Available data show that CDDs and CDFs can be (1) formed in the environment; (2) degraded; (3) remain unchanged; and (4) migrate through soil to ground water. The most useful predictive information comes from actual field measurements as well as laboratory experiments which have been constructed to simulate field conditions closely. The fairly sizable number of environmental fate experiments, especially in the area of light-related effects is confusing, but a general understanding of this fate is beginning to emerge.

Phototransformation

CDDs and CDFs resist sunlight-induced breakdown when they are present in water and on dry surfaces such as soil, wood, and glass (i.e., solid-phase surfaces). This resistance is increased with increasing number of chlorine atoms in the molecule. When chlorine atoms are lost under solid-phase conditions, those in the most toxic 2,3,7,8-substituted positions appear to be preferentially retained. This relative stability contrasts with the instability demonstrated in laboratory experiments. In these, CDDs and CDFs were dissolved in organic solvents, which enhance breakdown, and were irradiated with ultraviolet light. These conditions promote transformation to less toxic CDDs and CDFs. In the field, if organic solvents are present, they would enhance the transport of CDDs and CDFs through the soil out of range of sunlight effects. This is a current explanation for the finding of CDDs in California and Florida ground water. Until recently, CDDs and CDFs were thought to be immobile in soil, tightly bound to soil particles due to their low water solubility, and therefore not a threat to ground water.

Microbial Degradation

Unlike the sometimes marked degradative effect that microorganisms have on many compounds, CDDs either resist transformation or are only slowly degraded by microorganisms.

Sometimes, transformation compounds cannot be identified and therefore their toxicities cannot be estimated. One fungal species has been shown to degrade 2,3,7,8-tetraCDD in a nitrogen-limited culture. The usefulness of this fungus to transform CDD contaminated soil awaits evaluation. Literature on microorganism effects on CDFs is lacking, but these compounds probably show similar resistance to transformation.

Volatilization

The U.S. EPA has recently noted that volatilization is a likely fate for CDDs in aquatic environments. This contrasts with an earlier conclusion that volatilization probably was not an important process. It is consistent with a 1981 evaluation by the National Research Council of Canada: In simulating the fate of 2,3,7,8-tetraCDD in two model aquatic ecosystems, 100 percent was estimated to be lost through volatilization and none to photolysis or microbial degradation. The Research Council concluded in 1984 that despite a lack of data for CDFs, but by inference from CDD data, volatilization could play a role in environmental distribution for this class of compounds.

Little information is available to predict the stability and extent of distribution of CDDs and CDFs once they have evaporated from water and land surfaces. They can exist in vapor and adsorb to particulate matter in air. The presence of CDDs and CDFs in lake sediments located on a Lake Superior island indicates that these compounds can be atmospherically transported and subsequently redeposited.

Persistence and Movement in Soils and Sediments

As noted above, CDDs and CDFs have been detected in ground water, probably by being transported through soil by organic solvents. CDDs have reached a depth of 30 meters in Florida, and 16 meters in California. In the absence of organic solvents, CDDs and CDFs are not expected to move downward to any great extent.

Migration of these compounds at waste disposal and land treatment sites cannot be predicted accurately by spiking solvent-free soil with CDDs and CDFs, and rinsing the soil with water. A standard soil leachate test specified by RCRA for dioxin-containing wastes requires use of water to leach CDDs from soil. A more accurate test would employ a mixture of water and organic solvents. The more accurate test would increase the amounts of CDD and CDF compounds extracted from soil and thereby their concentration in a leachate test. This in turn would increase the likelihood that CDD and CDF contaminated soil would not be acceptable under RCRA treatment standards. At present, the RCRA treatment standard requires that wastes found to contain any tetra-, penta-, or hexaCDDs or CDFs at concentrations of 1 ppb or more in a standard leachate test be treated before land disposal.

CDDs and CDFs are also expected to adsorb strongly to sediments and suspended particulate matter in water. As a result, and because of their stability, they are expected to be highly persistent in these associations. In aquatic systems, therefore, the major "sinks" for CDDs and CDFs will be sediments, suspended particulates, and biota.

Plant Uptake

Measurements of CDDs and CDFs have shown that these compounds are concentrated in aquatic plant extracts. This can be interpreted to show that CDDs and CDFs are taken up and concentrated by aquatic plants. However, an undetermined amount of this material may be adsorbed onto the plant surface rather than being absorbed by the plants. Bioaccumulation figures for these compounds in aquatic plants should be interpreted with some reservation, especially for unicellular phytoplankton where the surface area is large compared to the internal volume. The distinction is not important to zooplankton or fish consumers of aquatic plants:

adsorbed and absorbed CDDs and CDFs are both consumed with the food. With respect to terrestrial plants, EPA has recently concluded that 2,3,7,8-tetraCDD present in contaminated soil is "not likely" to concentrate in them. If true, plants would not be effective scavengers of CDDs and CDFs in soil, a use which has been suggested for on-site treatment of CDD and CDF-contaminated soil. Reported bioconcentration of CDDs by terrestrial plants may be due to contamination of leaf and plant surfaces by CDDs in dust and soil particles.

3. AQUATIC TOXICOLOGY

Two striking aspects of 2,3,7,8-tetraCDD toxicity to aquatic life are the (1) delayed toxic effects after brief periods of exposure; and (2) low concentrations which cause toxic reactions. Frequently, toxicity is not seen in the standard short-term, 96-hour acute test. Statistically significant adverse effects have been delayed for periods ranging from five to over 100 days after exposure to this chemical. Growth retardation is the most common effect reported for 2,3,7,8-tetraCDD. Other effects include fin necrosis, loss or underdevelopment of caudal fins, edema, liver necrosis, and hemorrhaging.

Toxic effects have been reported at water concentrations as low as 0.1 parts per trillion (ppt) 2,3,7,8-tetraCDD. The lowest acute LC_{50} value of 5.6 ppt for coho salmon is one order of magnitude lower than for two of the most toxic chemicals to aquatic life, endosulfan and toxaphene. (LC_{50} refers to the concentration of a chemical which kills 50 percent of a test population within a specified time period.)

Due to the delayed lethality normally found in 2,3,7,8-tetraCDD bioassays, the expression of LC_{50} for a 96-hour exposure is not a meaningful indicator of 2,3,7,8-tetraCDD toxicity. As a result, the literature concerning 2,3,7,8-tetraCDD describes modified LC_{50}s indicating mortality at some given time after the exposure period. There is no agreement on a standardized post exposure observation period for the calculation of LC_{50}.

Most toxicity studies with CDDs have focused on 2,3,7,8-tetraCDD. They have generally been short-term 96-hour exposures, and have been "static" or "static renewal" bioassays. The water and test chemical were either not renewed for the test period, or were renewed periodically as a batch replacement. Studying the toxicity of highly water insoluble compounds such as CDDs under static testing conditions can present difficulties. For example, a compound will tend to migrate out of the aqueous test solution and adsorb onto solid surfaces such as the test container, test organisms, or particulate debris. The adsorbed test chemical may not be available to the test organisms.

Adverse toxic reactions most likely would have been observed at lower concentrations of 2,3,7,8-tetraCDD than reported, if the bioassays had been the continuous-flow type. Here, both water and toxic chemicals are renewed on a continuing basis. This simulates many natural situations. Effects may be seen at lower water concentrations because of the continuous renewal of water containing the toxicant.

Few CDD chronic studies have been reported. CDF toxicity has been estimated only in studies where CDF-contaminated food was provided to the fish.

CDDs and CDFs accumulate in aquatic organisms. The highest reported bioconcentration factor for 2,3,7,8-tetraCDD is approximately 9,000 for both rainbow trout and mosquito larvae. This is possibly an underestimate of bioconcentration potential due to the static test condition.

One investigator exposed fish to a mixture of CDDs and CDFs containing from four to eight chlorine atoms. With few exceptions, those compounds having chlorines at the 2,3,7, and 8 positions were selectively concentrated by the fish. Others have observed that compounds with chlorine atoms in other positions also were accumulated by fish. In these latter experiments, the 2,3,7, and 8 compounds were not present. The extent to which molecular configuration influences uptake needs clarification.

Studies of elimination of CDDs and CDFs from fish that have been exposed to these compounds in water showed: (1) rate of elimination decreases with increasing chlorination of the compound; and (2) for the same degree of chlorination, CDFs are depurated at a greater rate than CDDs.

Subsequent to completion of this State Board report, data were published that showed higher toxicity and bioconcentration than previously reported. This new study, published in January 1988, described chronic effects of 2,3,7,8-tetraCDD and 2,3,7,8-tetraCDF on rainbow trout. The experiment was a 56-day flow-through test with 28 days of exposure followed by 28 days of depuration. At 38 parts per quadrillion 2,3,7,8-tetraCDD, the lowest concentration tested, significant adverse effects were observed on growth and survival. Because effects were determined at the lowest level, a no observed effect concentration (NOEC) for this CDD could not be derived. At 0.9 parts per trillion (ppt) 2,3,7,8-tetraCDF, reduced growth effects were reported and reduced survival was observed at 4 ppt. NOEC values were 0.4 ppt for growth and 1.8 ppt for survival for this CDF. While the

higher concentrations tested caused mortality within 28 days, the toxic effect of lower concentrations was not manifested until later. During the 28-day depuration period, mortality continued and there was no observed recovery in clean water.

The same study also reported bioconcentration factors of 39,000 for 2,3,7,8-tetraCDD and 6,049 for 2,3,7,8-tetraCDF. This newly published study concluded that 2,3,7,8-tetraCDD is more than 10,000 times as toxic to fish as the insecticides endrin or toxaphene and that 2,3,7,8-tetraCDF is roughly 1,000 times as toxic.

4. MAMMALIAN TOXICOLOGY

Absorption, Tissue Distribution, Metabolism, and Half-Lives

Both CDDs and CDFs are absorbed and concentrated by laboratory animals and humans. Up to 90 percent of the chemicals will be absorbed if they are present in food. Approximately 40 percent can be absorbed after skin application to laboratory animals.

The half-life of 2,3,7,8-tetraCDD in a 42 year old human volunteer was estimated to be 5.8 years. This is longer than the half-life of about one year for the same compound estimated for monkeys. It contrasts with 10 to 40 day half-lives measured in several small laboratory animals. Based on blood sample analyses, a half-life of greater than one year was calculated for 2,3,4,7,8-pentaCDF and 1,2,3,4,7,8-hexaCDF compounds in humans. These people had ingested rice oil contaminated with these and other CDFs in Taiwan. In Japan, following a similar incident, the same pentaCDF could still be detected in human blood 11 years after exposure.

Studies with 2,3,7,8-tetraCDD contaminated soil show that ingested soil can influence toxicity. Soil from a Times Beach, Missouri, area which was contaminated with waste oil containing CDDs and CDFs, produced a variety of adverse effects, including acute toxicity in laboratory studies. In contrast, contaminated soil from a 2,4,5-T and 2,4-D formulation site in New Jersey produced no toxicity in laboratory animals. Bioavailability of the chemicals, including CDDs and CDFs, appears to account for the difference between these two observations. This was estimated to range from 0.5 to 21 percent for the New Jersey soil and 25 to 85 percent for the Times Beach soil. Bioavailability refers to the amount which is expected to be absorbed into the animal's bloodstream and not tightly bound to the soil particles which would be eliminated as waste.

CDDs and CDFs can be expected to be distributed in the body in proportion to the amount of fat content of a particular tissue. In both laboratory animals and humans, highest concentrations are found in adipose tissue and liver.

Laboratory experiments with 2,3,7,8-tetraCDD and a CDF mixture have shown that these chemicals can move through the placenta. One study also showed that CDFs are transferred to the offspring in greater amounts through milk, compared to transport through the placenta.

Laboratory studies have shown that animals can transform absorbed 2,3,7,8-tetraCDD. Unidentified transformation products have been detected principally in the bile and urine. Depending on the compound, metabolites can be either more or less toxic than the parent from which they are derived. EPA has noted that metabolism of 2,3,7,8-tetraCDD appears to be mostly a detoxification process which produces metabolites less toxic than the parent compound.

Acute, Subchronic, and Chronic Toxicity Effects

As noted, one of the most acutely toxic substances known is 2,3,7,8-tetraCDD. However, species sensitivity can differ significantly. The male hamster is approximately 8000 times less sensitive than the male guinea pig in a short-term lethal dose test. When 2,3,7,8-tetraCDD is fed to animals in acutely toxic doses, death is delayed and may take from 5 to 45 days. During this period, weight loss occurs with the animals exhibiting a characteristic "wasting away" appearance. This compound also induces liver damage in most species. The immune system is adversely affected in all species tested. Thymic atrophy is the principal change. The spleen, lymph nodes, and bone marrow may be affected. Susceptibility to bacterial infection is increased, and antibody production decreased.

One experiment focused on the relative effects of technical grade pentachlorophenol (PCP) and its contaminants on immunosuppresion. The contaminants included chlorinated diphenyl ethers, phenoxy phenols, dibenzodioxins and dibenzofurans. Technical grade PCP contained 86 percent pentachlorophenol. This produced a dose-related decrease in antibody response. In contrast, analytical grade PCP, which was greater than 99 percent pure, had no effect. Neither did the chlorinated phenoxy phenol or diphenyl ether components. The experimenters concluded that a significant amount of the immunosuppression was caused by the CDDs and CDFs.

Most human exposures to CDDs and CDFs have occurred either occupationally or accidentally, and concurrently with exposure with other chemicals. In these situations the actual dose

received could not be determined. The most common human effects attributed to 2,3,7,8-tetraCDD exposures include chloracne, liver abnormalities, hematologic disorders, porphyria, and hyperpigmentation disorders. Also reported have been peripheral and central neurological disorders, lethargy and sensory impairment. Chloracne is characterized by comedones and cysts. These may subside within a few months or persist for years, with some cases reported lasting up to 15 years after exposure.

Other human exposure sources to 2,3,7,8-tetraCDD include (1) dirt roads in Missouri sprayed with waste oil containing 2,3,7,8-tetraCDD, and (2) Agent Orange, the herbicide used in Vietnam also contaminated with this compound. The 2,3,7,8-tetraCDD concentrations in the Missouri soil ranged from 39 to 2200 ppb. Persons exposed to this material had lived in the area from one to five years during the period of contamination. Signs of altered liver function included lower serum bilirubin and elevated urinary uroporphyrin concentrations. However, these measurements were considered to be "subclinical"; i.e., not significantly differing from the normal.

The Agent Orange exposure is discussed in the section "Carcinogenicity" below.

The Japanese rice oil contaminated with 1,000 ppm PCBs and 5 ppm CDFs, which included 0.45 ppm 2,3,7,8-tetraCDF, produced the following toxic effects in humans, collectively known as "Yusho": pigmentation disorders, chloracne, eye discharge, swelling of upper eyelids, distinctive hair follicles, and neurological disturbances.

Teratogenicity and Reproduction

2,3,7,8-TetraCDD is a teratogen to laboratory animals. Cleft palate is the most common malformation observed in mice. Kidney defects are also common as well as embryo toxicity. In rats, teratogenic effects include subcutaneous edema, hemorrhage in the gastrointestinal tract, kidney malformation, cleft palate, and vertebral defects. In monkeys there are insufficient data to clearly define a teratogenic response, although fetotoxicity has been observed. Studies of humans exposed to 2,3,7,8-tetraCDD in the chemical industry, during the Vietnam war and in forestry operations, have not been able to show a teratogenic or other adverse effect on reproduction. The animal data conclusively demonstrate that 2,3,7,8-tetraCDD is teratogenic and fetotoxic at low levels of exposure. They indicate a need to determine more carefully the potential for adverse human reproductive effects.

Studies whose purpose has been to determine the mutagenic potential of CDDs and CDFs have produced conflicting results. One of the reasons for this, at least for 2,3,7,8-tetraCDD, is that its high toxicity may preclude demonstration of a mutagenic response.

Carcinogenicity

Both 2,3,7,8-tetraCDD and a mixture of two hexaCDDs are potent animal carcinogens, as noted. At this time, although many people have been regularly exposed to CDD-contaminated formulations, there is little conclusive evidence linking CDD to human cancers. The difference between laboratory and human observations is surprising.

Public Law 96-151, enacted in December 1979, mandated the U. S. Veterans Administration to perform a comprehensive review and analysis of the world literature on Agent Orange and other phenoxy herbicides. Output from the original task has continued as a series of publications with Volumes IX and X being published in May 1987. These latest analyses show some associations between exposure to phenoxy herbicides, which may or may not have contained dioxins, and adverse human health impacts. However, the cited studies are noted to have shortcomings which "limit their usefulness as evidence of a cause-and-effect relationship." These include negative findings in observations made by other researchers and lack of ability to correlate effect with known exposure dose, or even to determine conclusively that all affected persons were exposed to the herbicide. One recent observation that needs further study is a statistically significant excess of non-Hodgkin's lymphoma in U. S. Marine Corps veterans who served in Vietnam compared to those who did not serve in Vietnam.

5. CRITERIA, STANDARDS, AND REGULATIONS

In the United States and Canada, criteria have been developed for certain chlorinated dibenzodioxins but not chlorinated dibenzofurans. The CDDs identified are 2,3,7,8-tetraCDD and "hexaCDD". The only agency to have adopted criteria as legally enforceable standards is the New York State Department of Environmental Conservation. The standards are for 2,3,7,8-tetraCDD: (a) 1 part per quadrillion in ambient water (10^{-6} ug/l); and (b) 35 parts per quadrillion in ground water (3.5×10^{-5} ug/l). The former is lower because of potential for bioaccumulation by aquatic organisms.

EPA has developed several criteria for 2,3,7,8-tetraCDD including those for the following: (1) ambient water for drinking purposes only (0.2 parts per quadrillion); (2) ambient water based on

consumption of fish and shell-fish only (0.014 parts per quadrillion); (3) ambient water based on consumption of water, fish and shellfish (0.013 parts per quadrillion); (4) total intake from all sources for humans (0.006 picograms per kilogram body weight per day); and (5) ambient air (0.03 picograms per cubic meter). Specific criteria are listed which relate to the one increased incidence of cancer per one million population risk level.

Other 2,3,7,8-tetraCDD criteria have been developed by the following agencies: (1) Michigan Department of Public Health (10 ppt in fish); (2) California Air Resources Board and Department of Health Services (30 femtograms per cubic meter in air); (3) U. S. Centers for Disease Control (1 ppb in soil); (4) U. S. Food and Drug Administration (50 ppt in fish); and (5) Ontario Ministry of the Environment, Ontario, Canada (20 ppt total intake from all sources for humans): The U. S. Food and Drug Administration also set an additional advisory level for consumption of fish containing 25 to 49 ppt 2,3,7,8-tetraCDD. Fish with concentrations in this range should not be consumed more than twice per month.

HexaCDD criteria have been developed by the following: (1) EPA (5.5 parts per quadrillion in drinking water; 0.8 picograms per cubic meter in air; and 0.16 picograms per kilogram body weight per day for all sources in humans); (2) California Air Resources Board and Department of Health Services (1 picogram per cubic meter in air); and (3) National Research Council of Canada (13 ppt in ambient water for human consumption of fish; and 20 ppt for fish flesh).

Regulations have been developed for both CDDs and CDFs which relate primarily to treatment methods and disposal. The California Department of Health Services regulates 2,3,7,8-tetraCDD in wastes disposed to land to protect against migration to surface and ground water. EPA has developed CDD and CDF treatment standards and prohibits land disposal of certain wastes containing these compounds unless treatment standards are achieved. The designated wastes include several chemicals with which CDDs and CDFs are associated as contaminants and include tri-, tetra-, and pentachlorophenol; tetra-, penta-, and hexachlorobenzene, and 2,4,5-T. They also include residues resulting from incineration or thermal treatment of soil contaminated with certain EPA-designated hazardous wastes. In addition, EPA regulations require registrants of pentachlorophenol to reduce the concentration of hexaCDD in three phases. By February 2, 1989, the maximum batch hexaCDD concentration allowed will be 4 ppm, with a maximum average of 2 ppm; this is a decrease from the present allowable maximum batch concentration of 15 ppm.

6. WOOD TREATMENT PRACTICES AND CALIFORNIA SITE CONTAMINATION

Chlorophenols such as pentachlorophenol (PCP), tetrachlorophenol (TCP), and their potassium and sodium salts, creosote, coal tars, and copper arsenate compounds have been used routinely at sawmills and wood treatment facilities in California. Wood is typically treated by immersing it in tanks containing the preservative solution, by spraying, or by forcing the solution under pressure into the wood to saturate the cells more fully for a longer lasting protection. Over time there is an accumulation of chemical residuals in sediments and sludge of the treatment systems. Often the treating, sorting, and drying areas become contaminated by the preservative solution.

Chlorophenols are recognized to contain CDDs and CDFs. The used preservative solution, including accumulated sediment and sludge, and contaminated soil, also contain CDDs and CDFs. Currently these wastes must be either stored on-site or disposed of outside of California because CDD and CDF-containing wastes are no longer accepted at California landfills. A nationwide ban on land-filling of dioxin-containing wastes goes into effect November 9, 1988.

On-site methods of disposal have been attempted; none are effective. These include burning in a teepee burner which, because of relatively low temperature burning, not only does not destroy CDDs and CDFs, but also produces them from precursor chlorophenol compounds. In addition, this procedure releases them to the environment adsorbed to the soot. Burial of wastes on-site also has been a common practice. As a temporary measure, on-site storage and containment of these materials in drums has been recommended as an interim disposal practice, but a long-term solution is still needed.

Three examples of California contamination occurring as a result of wood treatment operations are described. Each of these is in a different stage of the evaluation and cleanup process. They are representative of several additional sites in the state which are awaiting further investigation.

Oroville Wood Treatment Site: A 200-acre wood treatment facility near Oroville, Butte County, has been associated with the lumber industry since about 1920. Both PCP and creosote have been found in soil and ground water, both on and off-site. PCP in concentrations of up to 15,000 ppb has been detected in ground water below the site. (The California Department of Health Services Drinking Water Action Level for PCP is 30 ppb.) The depth to water is approximately 30 feet. A plume of PCP in concentrations up to 2000 ppb has been detected at least two miles south of the site. The depth to water in this area is

90 to 120 feet. Approximately 30 domestic wells have been found contaminated with PCP. No CDDs or CDFs have been detected. Residents have complained of various adverse health effects. A comprehensive study is underway to define the extent of PCP contamination.

Selma Wood Treatment Site: An 18-acre wood treatment facility has been in operation since approximately 1936 near Selma, Fresno County. As with similar facilities, a number of preservative chemicals have been used here including chromated copper arsenate and pentachlorophenol dissolved in a variety of solvents. Wastes were discharged into dry wells, into an unlined pond, as runoff into drainage ditches, to open ground, and into a sludge pit. PCP has been detected on-site in surface and ground water and in soil. Surface water concentrations have ranged from 0.24 to 2.3 ppm. The PCP ground water concentration was determined to be 2 ppb. The depth to water is approximately 30 feet. All tetra-through-octaCDD and CDF isomer groups, except tetraCDD, were detected in soil.

Off-site migration may have occurred since the vertical and horizontal extent of soil and ground water contamination has not been defined. EPA is currently conducting a sampling program to clarify this uncertainty.

Visalia Wood Treatment Site: This facility, in Tulare County, had used PCP for electrical pole treatment from 1968 to 1980, when operations ceased. Ground water contamination was detected in 1973 and has been followed since then. Hexa-, hepta-, and octaCDDs and CDFs, and PCP have been detected in shallow and deep aquifers. These and pentaCDFs also were detected in soil. There were no pentaCDDs detected in soil. PCP was detected in monitoring wells 600 feet to the south of the site at concentrations ranging up to 37 ppm and 1600 feet to the southwest at concentrations up to 2 ppm. Creosote was found in these samples. Additional monitoring wells were constructed in 1984 and soil cores taken during this work were analyzed to provide information on the vertical distribution of PCP, creosote, CDDs, and CDFs.

Ground water has been pumped from the shallow aquifer to the City of Visalia wastewater treatment plant since 1975. The purpose has been to reduce contaminant concentrations and prevent further migration away from the site. Additionally, a bentonite-cement slurry wall has been built below the surface to inhibit down-gradient movement of the contaminants. The barrier surrounds the shallow aquifer beneath the site and extends from the surface to its lower boundary. PCP, creosote, CDDs, and CDFs were detected in both aquifers whose waters were discharged to the treatment plant. All of these compounds were detected also in plant

influent, effluent, and sludge with one exception: CDDs and CDFs were not detected in the plant effluent. Water from the deep aquifer is used by the City of Visalia for drinking water. Sludge from the treatment plant has been used as a soil amendment by farms and residents. CDFs have been detected in soil. In 1985 a pretreatment system was installed at the site to remove ground water contaminants before water transfer to the treatment plant.

7. CALIFORNIA WATER RESOURCES CONTROL BOARD STUDY

The study reported here originally was based on potential pentachlorophenol contamination of the environment. The focus was on its use by sawmills and wood treatment plants. Samples taken for analysis included aquatic invertebrates and fish, treatment site runoff, ground water, and soil. At that time, State Board staff considered that environmental contamination by chlorinated dibenzodioxins and dibenzofurans might be of equal significance. (They were known to be contaminants of chlorophenol formulations.) To test this hypothesis, 13 samples were taken from five sawmills and one wood treatment plant (Table 3, Section A). Sample types and numbers were as follows: soil (4), sludge (4), dip tank liquid (2), and commercial chlorophenol formulations (3). Analyses detected significant CDD and CDF concentrations. A decision was made to base the study on CDD and CDF presence in areas of sawmills and wood treatment plants. Chlorophenols would become the subject of another survey.

This initial work showed that tetra-, penta-, hexa-, hepta-, and octaCDDs and CDFs were present in all 13 samples, with one exception; tetraCDDs were detected only in wet and dry sludge samples from one sawmill and in one pentachlorophenate commercial product. The commercial chlorophenol and chlorophenate products were found to contain both tetra-chlorophenol and pentachlorophenol.

Analyses at this stage identified CDDs and CDFs in terms of "isomer groups", e.g., "tetraCDD", "heptaCDF". The analyses did not identify specific CDDs and CDFs, e.g., 2,3,7,8-tetraCDD. Determination of the exact position of the chlorine atoms requires a rigorous analytical procedure. As noted earlier, a total of 210 individual CDDs and CDFs possibly can occur.

After CDD and CDF presence was firmly established in the 13 samples, a decision was made to concentrate future work on the 15 CDDs and CDFs that were toxicologically most significant, i.e., the tetra, penta, hexa, and hepta-chlorinated compounds

TABLE 3

CALIFORNIA WATER RESOURCES CONTROL BOARD
CHLORINATED DIBENZODIOXIN AND DIBENZOFURAN STUDY

A. Preliminary Screening: Isomer Group Analyses

1. 5 Sawmills and 1 Wood Treatment Plant: 13 samples as indicated:

   a. Soil (4)
   b. Sludge (4)
   c. Dip tank liquid (2)
   d. Commercial formulations (3)

B. Phase I: Compound Specific Analyses

1. Sawmill A (Trinity County): 2 samples

   a. Commercial sodium pentachlorophenate
   b. Dip tank sludge

2. Sawmill B (Glenn County): 2 samples

   a. Wet dip tank sludge
   b. Dry mix tank sludge

C. Phase II: Compound Specific Analyses

1. Sawmill C (Humboldt County): 4 samples

   a. Commercial potassium tetrachlorophenate
   b. Dip tank liquid
   c. Dip tank sludge (2 samples)

2. Wood Treatment Plant (San Joaquin County): 4 samples

   a. "Bloom"
   b. "Commercial"--recycled treatment material
   c. Soil at retort
   d. Sump liquid

which have four of the chlorine atoms located in the 2,3,7, and 8 positions (Table 2). Three of the target compounds are potent carcinogens to laboratory animals: 2,3,7,8-tetraCDD and a mixture of 1,2,3,6,7,8- and 1,2,3,7,8,9-hexaCDD.

In order to estimate the concentration of these 15 compounds in complex mixtures, which also include many non-2,3,7,8-substituted CDDs and CDFs, three methods can be followed. The first two are fairly straightforward. They require little more time than that to determine isomer group concentrations. One of these methods assumes that all tetra through heptaCDDs and CDFs are chlorinated at positions 2,3,7, and 8. This procedure could greatly overestimate the significance of the 2,3,7,8-substituted compounds since there may be a far greater number of non-2,3,7,8-substituted compounds present. The second method assumes that all compounds within an isomer group are present in equal numbers; e.g., 2,3,7,8-tetraCDD is one of 22 possible compounds in the tetraCDD isomer group, and its concentration would be 1/22 of the total tetraCDD concentration detected. While simple in concept, this procedure could significantly underestimate or overestimate the toxicity of a CDD mixture, depending whether or not 2,3,7,8-tetraCDD was present. The third method identifies each 2,3,7,8 CDD and CDF in a potential mixture of 210 CDDs and CDFs and numerous other interferences. This approach represents state-of-the-art analytical chemistry for CDDs and CDFs. It was the course chosen for the study reported here.

The work proceeded in two phases. Phase 1 was directed at analyzing some of the previously collected samples which were shown to contain high concentrations of CDD and CDF isomer groups. Phase 2 was initiated with additional samples once 2,3,7,8-substituted CDDs and CDFs were identified in Phase 1. All samples analyzed are described in Table 3.

A brief summary of the analytical results follows. All data are described in detail in the accompanying report appendices.

CDDs: 2,3,7,8-Chlorinated compounds from all four target isomer groups (tetra through hepta) were detected in all 12 samples analyzed, with the following exceptions: 2,3,7,8-tetraCDD was detected in only one sample and 2,3,7,8-pentaCDDs were detected in five of 12 samples.

CDFs: 2,3,7,8-Chlorinated compounds from all target isomer groups were detected in the 12 samples analyzed, with the following exceptions: tetraCDFs were found in 9 of 12 samples, with pentaCDFs and hexaCDFs in 10 of 12.

The concentration of the 2,3,7,8-substituted compounds was calculated also as a percentage of the total CDD or CDF concentration for each isomer group. Depending on the sample and the isomer group, the proportion of the 2,3,7,8 compounds ranged from a few percent to greater than 80 percent of the total concentration of the respective isomer group. This finding was based on analysis of the environmental samples and the two commercial chlorophenate products.

All samples except for one dip tank solution contained at least 1,000 ppb total tetra-through-hepta 2,3,7,8-chlorinated CDDs and CDFs (Table 4; Total 2,3,7,8 CDDs and CDFs). The total concentration of tetra-through-hepta 2,3,7,8-chlorinated CDDs and CDFs ranged between 44 and 41,000 ppb. The concentrations of 2,3,7,8-tetraCDD and CDF are given separately because of the high toxicity of the former, and of the latter by analogy. The presence of 2,3,7,8-tetraCDF is particularly significant because of its close structural resemblance to 2,3,7,8-tetraCDD. The table shows that 2,3,7,8-tetraCDF was present in all 8 samples taken at sawmills, up to concentrations of 200 ppb.

The study data also show that the following 2,3,7,8-chlorinated CDD and CDF compounds are most likely to be found as a result of tetrachlorophenol and pentachlorophenol use at sawmills and wood treatment plants.

1,2,3,6,7,8-hexaCDD
2,3,7,8-tetraCDF
1,2,3,7,8-pentaCDF
2,3,4,7,8-pentaCDF
1,2,3,6,7,8-hexaCDF

Although the data are complex, a brief overview of analyses of these 12 samples indicates the following: 2,3,7,8-chlorinated CDDs and CDFs are present as contaminants at sawmills and wood treatment plants, often at significant concentrations.

8. HAZARD EVALUATION

Compound Detection

As noted, a major assumption was made that most of the toxicity in CDD and CDF mixtures is contributed by the 2,3,7,8-chlorinated compounds. Laboratories in the United States and Sweden participating in the State Board Study obtained analytical standards for the 15 most toxic 2,3,7,8-CDDs and CDFs. Often these had to be synthesized since they were not commercially available. Analytical procedures were developed and refined for their detection. When detected in a sample, a concentration for each 2,3,7,8-chlorinated CDD and CDF was determined for each of

TABLE 4

SUMMARY OF 2,3,7,8-SUBSTITUTED CDD AND CDF CONCENTRATIONS IN TWELVE COMPOUND-SPECIFIC ANALYSES (TETRA, PENTA, HEXA, AND HEPTA ISOMER GROUPS) - ppb[1/]

Sample	2,3,7,8-Tetra-CDD	2,3,7,8-Tetra-CDF	All 2,3,7,8-Chlorinated CDDs[2/]	All 2,3,7,8-Chlorinated CDFs[2/]	Total 2,3,7,8-CDDs & CDFs[2/]
Commercial Na-PCP, Sawmill A	0	201	34,751	6,540	41,291
Commercial K-TetraCP, Sawmill C	0	200	1,197	1,148	2,345
Sawmill Dip Tanks					
Sawmill A sludge	0	15	25,305	3,333	28,638
Sawmill B wet sludge	0	17	2,332	485	2,817
Sawmill B dry sludge	9.7	95	15,411	2,177	17,588
Sawmill C center sludge	0	54	1,092	560	1,652
Sawmill C corner sludge	0[3/]	65	1,161	574	1,735
Sawmill C liquid	0	2.0	18	26	44
Wood Treatment Plant-					
PCP "Bloom"	0	4.4	24,183	10,712	34,895
Recycled "Commercial"	0	0	6,715	726	7,441
Soil at Retort Mouth	0	0	1,618	169	1,887
Sump Liquid	0	0	8,684	69	8,753

1/ Average of samples split between two laboratories.

2/ Does not include octaCDD and octaCDF.

3/ Reported at 6.8 ppb by one laboratory but not confirmed by second.

the 12 samples. For each of the samples, the relative proportion of the 2,3,7,8-chlorinated CDDs and CDFs in each isomer group also was calculated.

Toxicity Evaluation

Toxicity information was available for only a few of the 2,3,7,8-chlorinated compounds. In order to overcome this deficiency, three methods were considered to determine total sample toxicity based on toxicity of the individual 2,3,7,8-compounds.

1. The simplest approach is to assign the same "toxic equivalency factor" to each 2,3,7,8-chlorinated tetra-, penta-, hexa-, and heptaCDD and CDF, i.e., assume they are all equally toxic. The toxic equivalency factor is multiplied by the concentration of each compound detected to yield a "relative toxicity concentration." All products are added together to estimate a "total relative toxicity concentration" for all CDDs and CDFs in each sample.

   This approach does not take into consideration the different toxicities of individual compounds. It can be justified on the basis of limited toxicity information for most of the 2,3,7,8-substituted compounds, taking into account that toxicity generally was high where it has been measured.

2. The California Department of Health Services currently favors an approach which is based solely on data provided by carcinogenicity bioassays. Only two toxic equivalency factors can be estimated with this scenario because only 2,3,7,8-tetraCDD and a mixture of two 2,3,7,8-chlorinated hexaCDD compounds have been tested for carcinogenicity. With this method, all other CDDs and CDFs are assigned one or the other of the two factors. As with the first approach just described, each factor is multiplied by the appropriate compound concentration to estimate a relative toxicity concentration for each compound. The products also are added to estimate a total relative toxicity concentration for all CDDs and CDFs.

3. The U.S. Environmental Protection Agency has developed toxic equivalency factors for the 2,3,7,8-chlorinated CDDs and CDFs by taking into consideration both carcinogenicity information and other toxic effects data, such as those relating to reproductive effects. These equivalency factors are listed in Table 5. EPA also considers toxicity of non-2,3,7,8-chlorinated CDDs and CDFs and assigns them factors. These are one to three orders of magnitude less than those for the respective chlorinated compounds. Relative toxicity concentrations and total toxicities are estimated using the same steps described for the first two approaches.

TABLE 5

TOXIC EQUIVALENCY FACTORS FOR 2,3,7,8-CHLORINATED DIBENZODIOXINS AND DIBENZOFURANS (SOURCE: BELLIN AND BARNES, 1986)

	Compound	Toxic Equivalency Factor[2/]
CDD		
	2,3,7,8-tetraCDD	1.0
	1,2,3,7,8-pentaCDD	0.5
	1,2,3,4,7,8-hexaCDD	0.04
	1,2,3,6,7,8-hexaCDD	0.04
	1,2,3,7,8,9-hexaCDD	0.04
	1,2,3,4,6,7,8-heptaCDD	0.001
CDF		
	2,3,7,8-tetraCDF	0.1
	1,2,3,7,8-pentaCDF	0.1
	2,3,4,7,8-pentaCDF	0.1
	1,2,3,4,7,8-hexaCDF	0.01
	1,2,3,6,7,8-hexaCDF	0.01
	1,2,3,7,8,9-hexaCDF	0.01
	2,3,4,6,7,8-hexaCDF	0.01
	1,2,3,4,6,7,8-heptaCDF	0.001
	1,2,3,4,7,8,9-heptaCDF	0.001

1/ Bellin, J., and D. Barnes, 1986. Interim Procedures for Estimating Risks Associated with Exposure to Mixtures of Chlorinated Dibenzodioxins and Diobenzofurans (CDDs and CDFs). Risk Assessment Forum, U. S. Environmental Protection Agency EPA/625/3-87/012. Washington, DC.

2/ Toxic Equivalency Factors are based on carcinogenicity and other toxicity data relative to that for 2,3,7,8-tetrachlorodibenzodioxin (2,3,7,8-tetraCDD).

A comparison of the total relative toxicity concentrations estimated by the three methods for each of the 12 samples, shows a difference of three orders of magnitude between them (Table 6). The most conservative, i.e. highest, concentrations are based on the sum of all tetra through hepta 2,3,7,8-chlorinated CDDs and CDFs which have been given the same toxic equivalency factor (Method 1). These concentrations were 5 to 30 times greater than those calculated by the California Department of Health Services procedure (Method 2). The latter, in turn, were 3 to 30 times higher than those calculated by the EPA approach (Method 3). For example, the total relative toxicity concentrations calculated by the three methods for the "Sawmill B" dry-sludge sample were 17,588 ppb, 1094 ppb, and 330 ppb, respectively.

The authors of this report recommend that, until more 2,3,7,8-chlorinated compound-specific toxicity information is available, the EPA procedure be used to estimate the total relative toxicity concentrations of CDD and CDF mixtures. This method, unlike the previous two, takes into account all available toxicity information for the various CDD and CDF compounds.

Comparisons of relative toxicity concentrations also were made between CDD and CDF "isomer groups" in each sample for the 2,3,7,8-substituted compounds. In one sawmill dip-tank sludge sample, the compounds contributing the most relative toxicity, based on the EPA method, were the pentaCDFs (38 percent); the hexaCDDs (32 percent); and 2,3,7,8-tetraCDF (23 percent). These figures are based on a total sample relative toxicity concentration for CDDs and CDFs of 27.9 ppb (Sawmill C, Table 6).

The relative toxicity concentration of all 2,3,7,8-substituted CDFs in the same sample was approximately twice that of the CDDs. The estimated relative toxicity concentration for these CDFs was 18 ppb and for CDDs, 9.9 ppb.

Future Sample Toxicity Evaluation

The authors recommend a simplified approach to estimating total CDD and CDF toxicity of similar samples in future analyses. It is based on (1) performing isomer group analyses; and (2) using the ratios of the 2,3,7,8-chlorinated compounds identified in this study, relative to isomer group concentrations. These ratios can be used to estimate relative toxicity concentrations for similar sample types, when only isomer group analyses are performed. The current data bases (12 samples) can be increased by additional compound-specific analyses by other specialist laboratories. CDD and CDF isomer group analyses can be performed by many commercial laboratories. Only a few laboratories in the United States are capable of doing the more definitive analyses on a reasonable schedule.

TABLE 6

TOTAL RELATIVE TOXICITY CONCENTRATIONS (ppb) OF 2,3,7,8-CHLORINATED DIBENZODIOXINS AND DIBENZOFURANS: A COMPARISON BASED ON THREE METHODS[1/]

	Method 1	Method 2	Method 3
SAMPLE	TEF=1[2/]	CDHS 1986[3/]	Bellin and Barnes, EPA 1986[4/]
Commercial Na-PCP			
Sawmill A	41,291	2,055	289.5
Commercial K-tetraCP			
Sawmill C	2,345	463	72.8
Sawmill Dip Tanks			
Sawmill A sludge	28,638	1,184	139.1
Sawmill B wet sludge	2,817	173	32.0
Sawmill B dry sludge	17,588	1,094	329.6
Sawmill C center of tank sludge	1,652	216	27.0
Sawmill C corner of tank sludge	1,735	218	27.9
Sawmill C liquid	44	8.4	0.8
Wood Treatment Plant-PCP			
"Bloom"	34,895	1,120	100.5
Recycled "Commercial"	7,441	223	11.3
Soil at Retort Mouth	1,887	64	5.6
Sump Liquid	8,753	274	9.8

1/ OctaCDD and octaCDF were not considered in the calculations due to estimated low toxicity.

2/ Toxic Equivalency Factor = 1 for each 2,3,7,8-chlorinated CDD and CDF.

3/ California Department of Health Services, 1986; Relative potency of 2,3,7,8-tetra- and pentaCDDs and CDFs = 2,3,7,8-tetraCDD; and 2,3,7,8-hexa- and heptaCDDs and CDFs = 2,3,7,8-hexaCDD (or 0.03 2,3,7,8-tetraCDD).

4/ Bellin, J. and D. Barnes. 1986. Interim Procedures for Estimating Risks Associated with Exposure to Mixtures of Chlorinated Dibenzodioxins and Dibenzofurans (CDDs and CDFs). Risk Assessment Forum, U. S. Environmental Protection Agency EPA/625/3-87/012. Washington, DC.

The only site-specific cleanup level that has been established for CDDs or CDFs in the United States has been 1 ppb for 2,3,7,8-tetraCDD in Times Beach, Missouri. Total relative toxicity concentrations calculated for the 12 samples in this study -- using the EPA method -- showed that 11 exceeded 1 ppb. These ranged from 5.6 to 329.6 ppb (Table 6).

The concentration for the twelfth sample was 0.8 ppb. All 12 exceeded the 1 ppb level based on the California Department of Health Services' method of calculation.

Setting a Clean Up Level. Contamination by CDD and CDF mixtures associated with chlorophenol products used at sawmills and wood treatment plants should be cleaned up following a site-specific procedure. The present study concludes that the DHS California Site Mitigation Decision Tree Manual, although complex, should be followed. The Decision Tree includes five components:

1. Preliminary risk appraisal;
2. Site assessment;
3. Risk appraisal;
4. Environmental fate and risk determination; and
5. Determination of mitigation strategy and remedial action plan selection.

The risk appraisal phase uses applied action levels (AAL) for specific media of exposure such as air, soil, water, and biota. These have been set to protect specific biological "receptors". The AALs also take into account the amount of a substance taken in by inhalation, ingestion, and adsorption, as well as other toxicological factors such as absorption, metabolism, distribution, and elimination characteristics of the medium.

The California Department of Health Services is currently reviewing a consultant's report containing proposed air and water AALs for CDDs. A CDHS report describes a strategy for developing AALs related to soil contact. Numerical AALs for CDDs in soil will be proposed by CDHS in 1988.

Characterization of CDD and CDF mixtures in samples by calculating total relative toxicity concentrations will allow an estimate of potential hazard. The options for remedial action can then be identified. At some sites, moving the material may create more of a hazard than encapsulation and on-site storage. On-site storage with material isolated from humans and the environment may be the most effective interim measure until acceptable methods of CDD and CDF destruction are available.

CHAPTER 1: INTRODUCTION

Why are polychlorinated dibenzo-p-dioxins (CDDs) and dibenzofurans (CDFs) important? The best known and most studied of the CDDs is the chemical 2,3,7,8-tetrachlorodibenzo-p-dioxin, commonly called "dioxin". In ranking the potency of 55 suspected human carcinogens, the U.S. EPA (1985b) listed "dioxin" as the most potent -- 50 million times more potent than trichlorethylene (TCE) or vinyl chloride. This CDD compound also is highly toxic in a single dose to certain animal species. In a single feeding of one part "dioxin" to one billion parts body weight, half of the guinea pigs dosed will die. However, unlike a lethal dose from many other highly toxic chemicals, death is delayed from 5 to 45 days after exposure occurs. In addition to 2,3,7,8-tetrachlorodibenzo-p-dioxin, several other CDDs and CDFs are of probable toxicological concern.

BACKGROUND

The California Water Resources Control Board's (State Board) investigation of chlorinated dibenzo-p-dioxins and dibenzofurans originated with a study of pentachlorophenol (PCP). This compound is a major industrial chemical and biocide used worldwide. In California, PCP has been used extensively for wood preservation at lumber mills and wood treatment plants. Typically, a water soluble form of PCP is used at sawmills for surface protection against fungal staining of lumber. In contrast, wood treatment plants inject insoluble PCP under pressure for long-term protection of materials such as poles and posts. Most of these facilities are located in two areas of the state, the northwest and the central valley. Investigations by the Regional Water Quality Control Boards and other agencies have documented a number of effects on California's environment. These include fish kills; contaminated soil, surface water and ground water; accumulation in marine sediments and organisms; and incidents of worker exposure. A few California studies have also detected CDDs and CDFs in both commercial PCP and PCP-contaminated soil.

In the past five years, conditions at sawmills have noticeably improved. Some mills have converted to systems that completely contain and recycle wood preservative chemicals on site, preventing environmental contamination. In particular, the "unit dip" tank has been successful. In a unit dip operation, a below ground rectangular tank is filled with a wood preservation solution (typically the soluble form of pentachlorophenol is diluted 1:100 parts water). Sawn lumber is bundled together, immersed in the tank, then allowed to dry in a covered building sloped such that drippage drains back into the dip tank. A side

effect of this "best management practice" is accumulation of sawdust and dirt on the tank bottom that forms a sludge. Eventually, the sludge becomes deep enough to interfere with dip operations and must be removed. An examination of two Swedish sawmills that used chlorinated phenols as wood preservatives noted that these sludges became "remarkedly enriched" in chlorinated dibenzofurans (Levin et al., 1976). Levels of total CDFs as high as 700 ppm were detected. Upon learning of the findings in Sweden, the State Board's priority chemical study of pentachlorophenol was expanded to include monitoring for CDDs and CDFs in dip tanks and other locations at sawmills and to investigate contaminant levels at wood treatment plants.

NOMENCLATURE OF CDDs AND CDFs

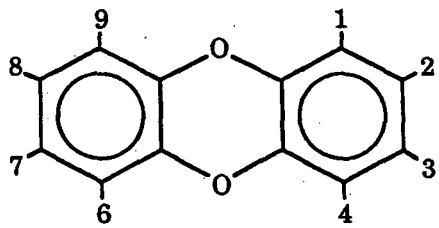
Although the term "dioxin" has become synonymous with 2,3,7,8-tetrachlorodibenzo-p-dioxin, "dioxin" is not used elsewhere in this document because the subject of this report is not one but a number of different CDDs. The nomenclature of CDDs and CDFs is important because there are enormous differences in toxicity between compounds. Those compounds chlorinated at the 2,3,7, and 8 positions and containing from four to seven chlorine atoms are believed to be most toxic. In this report, these CDDs and CDFs are referred to as 2,3,7,8 chlorine-substituted compounds or, more simply, as 2,3,7,8 congeners.

The basic skeleton of all the CDDs is dibenzo-p-dioxin, a molecule containing two benzene rings joined by two oxygen atoms (Figure 1.1). The dibenzo-p-dioxin molecule is chlorinated if a chlorine atom is attached to any of the positions numbered 1 through 4 and 6 through 9. The dibenzo-p-dioxin skeleton can accommodate up to eight chlorine atoms. 2,3,7,8-Tetrachlorodibenzo-p-dioxin contains four chlorine atoms, one each at the 2,3,7, and 8 positions (Figure 1.1). For purposes of simplification, 2,3,7,8-tetrachlorobdibenzo-p-dioxin is abbreviated as 2,3,7,8-tetraCDD in this document. Numbers indicate location of chlorine atoms on the molecule and tetra refers to four chlorines. Other four chlorine dibenzo-p-dioxins can also occur, for example 1,4,6,9-tetraCDD. In fact, there are 22 different ways that four chlorines can be arranged on the molecule; in chemical terminology there are 22 different "isomers".

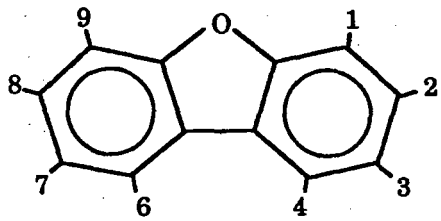
There are three terms of chemical nomenclature that are used in this document to characterize CDDs: isomer group, isomer, and congener. CDDs can be divided into eight groups called isomer groups (also called homologues), with each isomer group containing the same number of chlorine atoms. For example, tetraCDD is the four chlorine isomer group of CDDs. An isomer is defined by the arrangement of chlorine atoms within an isomer

FIGURE 1.1

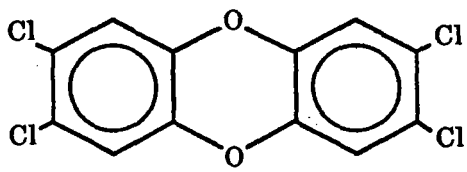
CHEMICAL STRUCTURES



Dibenzo-p-dioxin



Dibenzofuran



2,3,7,8-Tetrachlorodibenzo-p-dioxin
(2,3,7,8-TetraCDD)
("Dioxin")

group. The CDDs 1,4,6,9-tetraCDD and 2,3,7,8-tetraCDD are isomers within the tetraCDD isomer group because both CDDs contain 4 chlorines. The term CDD congener refers to any CDD compound. For example, the CDDs 2,3,7,8-tetraCDD and 1,2,3,7,8-pentaCDD both are highly toxic. They are both CDD congeners but are not isomers because one contains four chlorine atoms and the other has five chlorines. There are a total of 75 CDD congeners (Table 1.1).

Similar nomenclature refers to the dibenzofurans. Because there is only one oxygen atom in the skeleton (Figure 1.1), the molecule is less symmetrical, and there are more congeners due to a larger number of possible chlorine arrangements. For example, while there are 22 tetraCDD isomers, there are 38 tetraCDF isomers. There are a total of 135 congeners in the eight CDF isomer groups.

In this report, as stated above, 2,3,7,8 chlorine-substituted compounds also are referred to as 2,3,7,8 congeners. While public health and environmental concerns in the United States initially focused on 2,3,7,8-tetraCDD, there has been increasing awareness that presence of the other 2,3,7,8 congeners among the CDDs and CDFs should also be closely monitored.

The CDDs and CDFs have not been deliberately manufactured except for use as laboratory standards to confirm chemical analyses. Rather, these compounds appear as by-products of chemical synthesis, electrical equipment fires, municipal incineration of solid wastes and other causes (See Appendix B "Sources"). Although 2,3,7,8-tetraCDD has not been reported in pentachlorophenol formulations manufactured in the United States, other 2,3,7,8 CDD and CDF congeners are present in commercial PCP formulations.

CALIFORNIA WATER RESOURCES CONTROL BOARD STUDY

By late 1984, the State Board had sampled for presence of CDDs and CDFs at several sawmills with emphasis on soils, dip tank liquids, and sludges. The results showed that CDDs and CDFs were present where pentachlorophenol had been used for wood preservation. The chemical analyses were for isomer groups (isomer group analysis); concentrations were reported as 50 ppb "hexaCDD", for example, without identification of specific isomers within an isomer group. The State Board informed other agencies of the findings, and an interagency group subsequently met with the Secretary of Environmental Affairs. The consensus of the meeting was that the findings were provocative and of concern, but confirmation of results was necessary. The interagency group agreed with State Board staff that, if possible, future studies should attempt to: (1) validate results

TABLE 1.1

CDD AND CDF ISOMER GROUPS, ISOMERS, AND CONGENERS

Isomer Group	Number of Isomers in Isomer Group
CDDs	
1. Monochlorodibenzodioxin (monoCDD)	2
2. Dichlorodibenzodioxin (diCDD)	10
3. Trichlorodibenzodioxin (triCDD)	14
4. Tetrachlorodibenzodioxin (tetraCDD)	22
5. Pentachlorodibenzodioxin (pentaCDD)	14
6. Hexachlorodibenzodioxin (hexaCDD)	10
7. Heptachlorodibenzodioxin (heptaCDD)	2
8. Octachlorodibenzodioxin (octaCDD)	1
TOTAL CDD CONGENERS	75
CDFs	
1. Monochlorodibenzofuran (monoCDF)	4
2. Dichlorodibenzofuran (diCDF)	16
3. Trichlorodibenzofuran (triCDF)	28
4. Tetrachlorodibenzofuran (tetraCDF)	38
5. Pentachlorodibenzofuran (pentaCDF)	28
6. Hexachlorodibenzofuran (hexaCDF)	16
7. Heptachlorodibenzofuran (heptaCDF)	4
8. Octachlorodibenzofuran (octaCDF)	1
TOTAL CDF CONGENERS	135

by splitting samples between laboratories; and (2) determine if 2,3,7,8 CDD and CDF congeners were present in sawmill residues. This latter approach, referred to as congener-specific analysis, is difficult and represents state-of-the-art analytical chemistry. This report describes the results of subsequent 2,3,7,8 congener-specific analysis commissioned by State Board staff, as well as the results of the earlier isomer group analyses.

CHAPTER 2: ENVIRONMENTAL FATE

Despite considerable research, there are little reliable qualitative or quantitative data with which to predict the environmental fate of 2,3,7,8-tetraCDD accurately. Much less is known about other polychlorinated dibenzo-p-dioxins (CDDs) (U.S. EPA, 1985b) and even less about the polychlorinated dibenzofurans (CDFs) (NRCC, 1984). The bulk of observations in this chapter, therefore, concern 2,3,7,8-tetraCDD; inferences occasionally are made concerning the behavior of other CDDs and CDFs based on 2,3,7,8-tetraCDD behavior.

Sections of this chapter address five areas: (1) phototransformation; (2) microbial degradation; (3) volatilization; (4) persistence and movement in soil and sediment; and (5) plant uptake. Major emphasis is placed on phototransformations, as this area has been a subject of considerable research and some controversy.

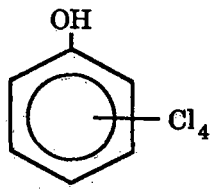
PHOTOTRANSFORMATION

The subject of CDD and CDF phototransformations has received considerable attention in recent years. Until recently, it was believed that these compounds would break down to less toxic compounds in the environment. However, it is now known that many of the conditions used in laboratory studies are not necessarily representative of environmental situations. Major differences are apparent depending on whether the photoreactions occur in organic solvents ("solution phase" reactions) or on surfaces such as wood or glass ("solid phase" reactions). Under solid phase conditions, it is possible for more toxic CDDs and CDFs to be produced from less toxic cogeners.

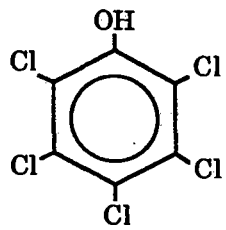
Polychlorinated dibenzo-p-dioxins (CDDs) and dibenzofurans (CDFs) have been shown to be both formed and degraded by sunlight (NRCC, 1984; U.S. EPA, 1985b). Precursor compounds are found in commercial chlorinated phenol wood preservative formulations and include chlorinated diphenyl ethers, "predioxins" (chlorinated-2-phenoxyphenols), and tetrachlorophenol and pentachlorophenol (Figure 2.1) (Norstrom et al., 1977; Nilsson et al., 1974; Choudhry and Hutzinger, 1982; Kitunen et al., 1987). Further, highly chlorinated CDDs and CDFs may be dechlorinated photolytically to produce more toxic CDDs and CDFs (Buser, 1976; U.S. EPA, 1985b). A summary of phototransformation reactions of CDDs and CDFs is given in Table 2.1, and described in more detail as follows:

FIGURE 2.1

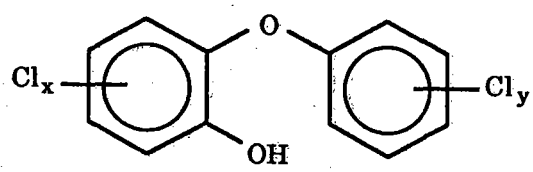
COMPOUNDS PRESENT IN COMMERCIAL FORMULATIONS OF CHLOROPHENOLS THAT MAY BE TRANSFORMED TO CDDS AND CDFS



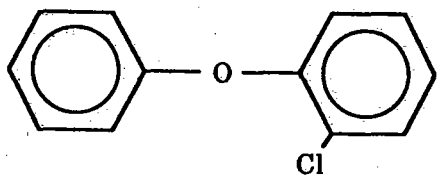
Tetrachlorophenol



Pentachlorophenol



"Predioxin"
Chloro-2-phenoxyphenol



Polychlorinated Diphenyl Ether (PCDE)
(2 - Chlorodiphenyl Ether)

TABLE 2.1

SUMMARY OF PHOTOTRANSFORMATIONS OF CDDs AND CDFs

Process	Medium	Compound or Precursor	Fate or Product	Reference
Formation Reactions				
1.	water,UV	Na-PCP	octaCDD	Crosby et al., 1973
2.	wood, sunlight	PCP	octaCDD	Cull and Dobbs, 1984
3.	wood, sunlight	purified PCP	octaCDD heptaCDD hexaCDD	Lamparksi et al., 1980
4.	methanol, UV	predioxins	tetraCDD triCDDs diCDD diCDF	Nilsson et al., 1974
5.	hexane UV	PCDE (polychlori-nated diphenyl ethers)	CDFs	Norstrom et al., 1976 and 1977
6.	methanol, UV	PCB (tetra-chloro-PCB)	CDF (diCDF)	Choudhry and and Hutzinger, 1982
Photolysis				
A. Solution Phase	organic solvents, UV or sunlight	higher CDDs or CDFs	lower CDDs or CDFs (preferential lateral chlorine removal)	Buser and Rappe, 1978
1.	benzene-hexane, UV	octaCDD	1,2,3,4,6, 7,9-heptaCDD (major product)	Buser, 1976

TABLE 2.1 (continued)

SUMMARY OF PHOTOTRANSFORMATIONS OF CDDs AND CDFs

Process	Medium	Compound or Precursor	Fate or Product	Reference
2.	benzene-hexane, UV	1,2,3,6, 7,8-hexaCDD	1,2,3,6,8-pentaCDD (major pentaCDD), 1,3,6,8-tetraCDD (major tetraCDD), 2,3,7,8-tetraCDD (trace)	Buser, 1979
3.	benzene-hexane, 310nm UV	1,2,3,7, 8,9-hexaCDD	1,2,3,7,9-pentaCDD (major pentaCDD), 1,3,7,9-tetraCDD (major tetraCDD), 2,3,7,8-tetraCDD (trace)	Buser, 1979
4.	methanol, 310nm UV	octaCDF	mixture of tetra to OctaCDF	Hutzinger et al., 1973
5.	hexane, 254nm UV	tetraCDFs	triCDFs	Mazer and Hileman, 1982
6.	methanol 310nm UV	2,8-diCDF	2-monoCDF	Crosby et al., 1973
7.	benzene-hexane, UV	octaCDF	heptaCDFs (all 4 isomers) hexaCDFs (13 of 16 possible isomers)	Buser, 1976

TABLE 2.1 (continued)

SUMMARY OF PHOTOTRANSFORMATIONS OF CDDs AND CDFs

	Process	Medium	Compound or Precursor	Fate or Product	Reference
B.	Solid Phase	wood, UV or sunlight	higher CDD	lower CDD (preferential peri chlorine removal	Lamparski et al., 1980
		wood, UV or sunlight	octaCDD	1,2,3,4, 6,7,8-heptaCDD (major product)	Lamparski et al., 1980
C.	Gamma irradiation	benzene-hexane, gamma irradiation	octaCDD octaCDF	lower CDDs and CDFs (non-preferential chlorine removal)	Buser, 1976

Formation Reactions

Crosby et al. (1973) reported that low levels of octaCDD were formed when CDD-free sodium pentachlorophenate in water was irradiated (Figure 2.2). Pentachlorophenol present on wood has been reported to be transformed by sunlight to octaCDD (Choudhry and Hutzinger, 1982). The production of octaCDD is of concern because it has the potential to be dechlorinated and form more toxic CDDs. Cull and Dobbs (1984) have suggested that, although there may be a short-term increase in CDD levels when wood treated with PCP is exposed to sunlight, in the long-term, CDD photolytic breakdown and/or volatilization will counter CDD formation reactions. They found no evidence of increased concentrations of any CDD congeners on treated wood exposed to sunlight for periods of up to 30 months.

Lamparski et al. (1980) conducted a series of experiments with natural and artificial sunlight in which they irradiated pentachlorophenol-treated wood and observed octaCDD and hexaCDD concentrations. Initially, in wood treated with purified pentachlorophenol (contaminants removed), levels of octaCDD increased; after 10 to 20 days, octaCDD levels stabilized. According to Lamparski et al. (1980), the hexaCDDs and heptaCDDs measured after irradiation were formed by the dechlorination of octaCDD rather than by pentachlorophenol condensation. The authors concluded that at least two reactions were occurring: formation of octaCDD from pentachlorophenol and degradation of octaCDD to lower chlorinated CDDs.

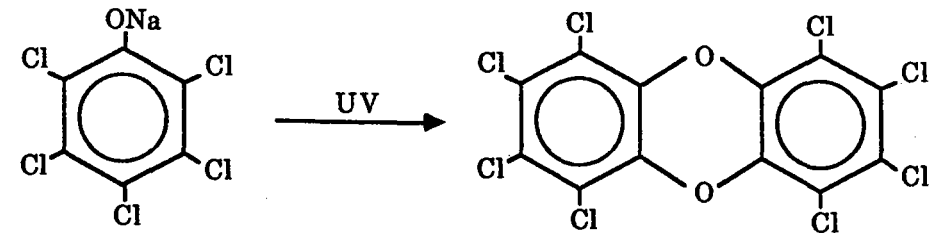
Polychlorinated phenoxyphenols (PCPPs) occur as major contaminants in chlorophenols (Humppi et al., 1984). The PCPPs containing a hydroxyl at the 2 position (Figure 2.1) are referred to as "predioxins" (chlorinated-2-phenoxyphenols) that have been reported to be the main two-ringed impurity in 2,4,6-trichlorophenol, 2,3,4,6-tetrachlorophenol, and pentachlorophenol (Nilsson et al., 1974). Predioxins have been shown to undergo photochemical ring closure to form CDDs (Nilsson et al., 1974). Nilsson et al. (1974) found that one tetraCDD, two triCDDs, one diCDD, and one diCDF were formed from photolysis of a methanol solution of pentachloro-2 phenoxyphenol (Figure 2.2), and concluded that predioxins "are easily metabolized" to CDDs and CDFs "under environmental conditions." However, as noted elsewhere, methanol provides a rich hydrogen-donor source, and laboratory experiments may not predict the behavior of predioxins in the environment.

In a review of CDF phototransformation studies, Choudhry and Hutzinger (1982) have reported formation of CDFs from polychlorinated diphenyl ethers (PCDEs) irradiated in organic

FIGURE 2.2

CDD AND CDF FORMATION REACTIONS

A. PCP to OctaCDD

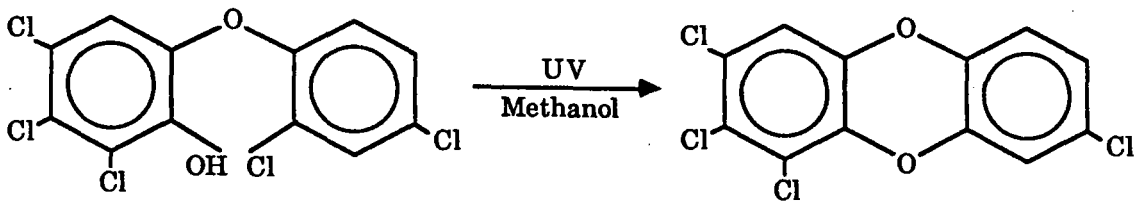


Sodium Pentachlorophenate

Octachlorodibenzo-p-dioxin

(Adapted from Crosby et al., 1973)

B. Predioxin to CDD

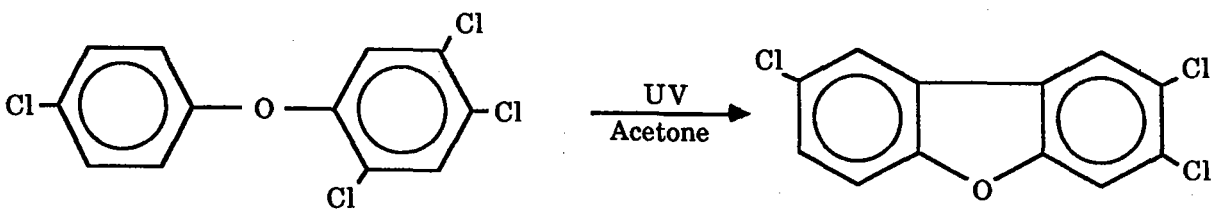


4,5,6-Trichloro-2-(2,4-dichlorophenoxy) phenol
"Predioxin"

1,2,3,8-TetraCDD

(Adapted from Nilsson et al., 1974)

C. Chlorinated Diphenyl Ether to CDF



2,4,4',5-Tetrachlorodiphenyl Ether

2,3,8-TriCDF

(Adapted from Choudhry and Hutzinger, 1982)

solvents (Figure 2.2) and from polychlorinated biphenyls (PCBs) irradiated in water. The PCDEs, which have been detected in technical grade chlorinated phenols at levels as high as 100 ppm (Norstrom et al., 1977), required at least one chlorine to be present in the 2 position (Figure 2.1) for CDF formation to occur (Choudhry and Hutzinger, 1982). Norstrom et al. (1976) noted that formation of CDFs from PCDEs "can be a reaction of environmental significance" and recommended minimizing PCDE levels in commercial chlorinated phenols.

PCBs have been detected in widely distributed aquatic environments and, because PCBs and other highly hydrophobic compounds appear to accumulate in organic films as a surface microlayer in natural waters, there is a potential for CDF formation (Choudhry and Hutzinger, 1982). CDFs have also been detected as contaminants of PCBs produced in several countries (Bowes et al., 1975).

The formation of CDDs and CDFs by dechlorination of more highly chlorinated CDDs and CDFs (Figure 2.3) is discussed in the following section.

Photolysis

CDDs

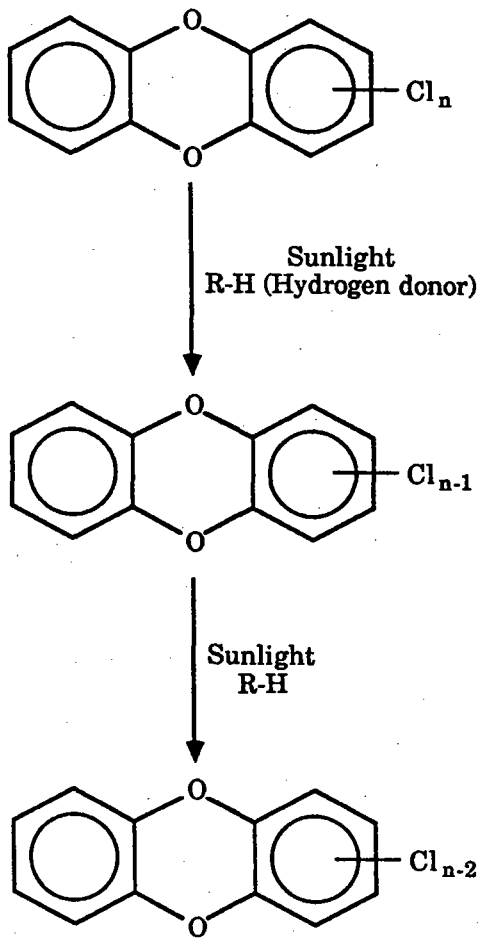
A number of studies have examined various aspects of CDD photolytic breakdown. While most experiments have used hydrogen-donating organic solvents (solution phase), some have examined CDD photolysis on solid surfaces such as wood or glass (solid phase). As discussed below, there is evidence that different breakdown products occur in solutions compared to reactions on solid surfaces (Lamparski et al., 1980; Choudhry and Hutzinger, 1982). The rates of CDD degradation also change depending on the medium. Thus, direct extrapolation of photolytic results in the laboratory to events in the environment, particularly where hydrogen-donating materials may not be present, is probably not warranted.

For photolytic degradation of CDDs and CDFs to occur, certain conditions must be met. Crosby (1985) noted that early studies of 2,3,7,8-tetraCDD suggested that the compound was not readily photolyzed by sunlight. He noted that there are three requirements for effective photolysis of 2,3,7,8-tetraCDD in a medium:

1. incident light must contain the UV wavelengths absorbed by the compound,

FIGURE 2.3

PHOTOLYTIC DECHLORINATION
(From NRCC, 1981)



2. light must penetrate the medium, and

3. hydrogen-donors must be present.

These three requirements were not met in earlier studies because:

1. studies were conducted in greenhouses (UV light filtered out by glass),

2. no degradation was observed in soil (no UV penetration beneath soil surface layer), or

3. crystalline films of 2,3,7,8-tetraCDD were exposed in glass dishes (no hydrogen-donor).

In general, the more highly chlorinated CDDs are more resistant to photodegradation (Crosby et al., 1971). For example, octaCDD is less reactive than triCDD. However, the positioning of chlorine atoms on the molecule is very important (U.S. EPA, 1985a). Buser and Rappe (1978) reported a preferential loss of chlorine atoms at the 2,3,7, and 8 positions in higher CDDs. In summarizing data on UV photolysis of octaCDD in organic solvents, Buser and Rappe (1978) reported that the major heptaCDD formed was the 1,2,3,4,6,7,9-substituted compound (Figure 2.4). With continued photolysis, the major hexaCDD appeared to be either 1,2,4,6,7,9-hexaCDD or 1,2,4,6,8,9-hexaCDD. The same congeners were obtained by irradiating octaCDD with sunlight. Dobbs and Grant (1979) measured half-lives of three hexaCDDs irradiated by sunlight in hexane solution and reported 5.4 hours for 1,2,3,7, 8,9-hexaCDD, 17 hours for 1,2,3,6,7,9-hexaCDD, and 47 hours for 1,2,4,6,7,9-hexaCDD.

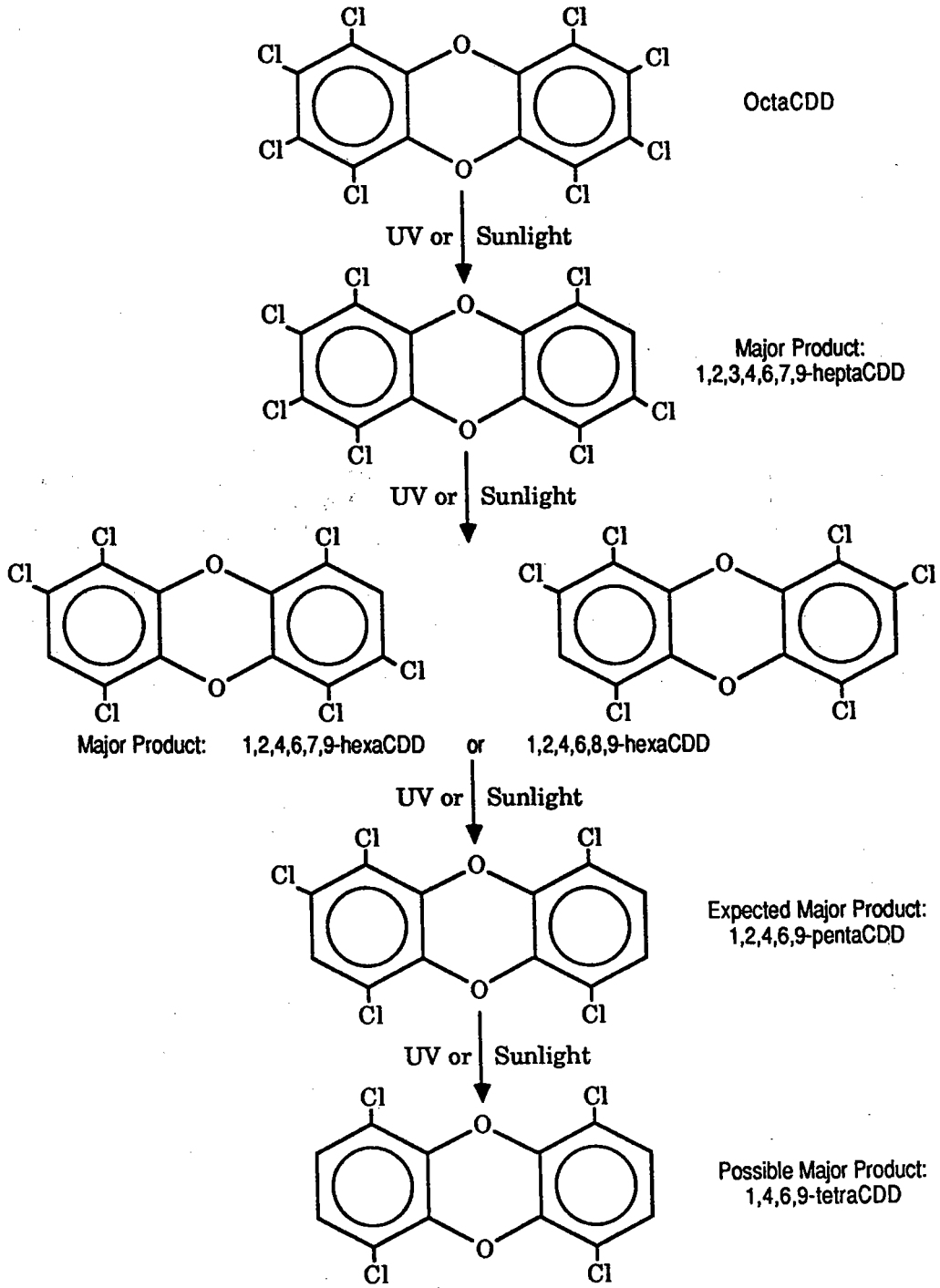
Buser (1979) dissolved two hexaCDD isomers, 1,2,3,6,7,8-hexaCDD and 1,2,3,7,8,9-hexaCDD, separately in a mixture of 5 percent benzene in n-hexane. The hexaCDD isomers were irradiated with UV and the dechlorination products determined. Buser (1979) reported a preferential removal of lateral chlorines: the major product of 1,2,3,6,7,8-hexaCDD was 1,2,3,6,8-pentaCDD and for 1,2,3,7,8,9-hexaCDD, the major pentaCDD isomer was 1,2,3,7,9-pentaCDD. Examination of the tetraCDD isomers formed indicated that 1,3,6,8-tetraCDD was the major tetraCDD product from photolysis of 1,2,3,6,7,8-hexaCDD and 1,3,7,9-tetraCDD the major tetraCDD from 1,2,3,7,8,9-hexaCDD. Buser (1979) also reported trace amounts of 2,3,7,8-tetraCDD formed from both hexaCDD isomers.

As reported by U.S. EPA (1985b), Kim et al. (1975) found that photolytic dechlorinations of 1,2,3,6,7,8-hexaCDD and 1,2,3,7,8,9-hexaCDD produced only 13 percent of the toxic

FIGURE 2.4

PHOTOLYTIC DECHLORINATION OF OctaCDD (SOLUTION PHASE)

(Data from Buser and Rappe, 1978)



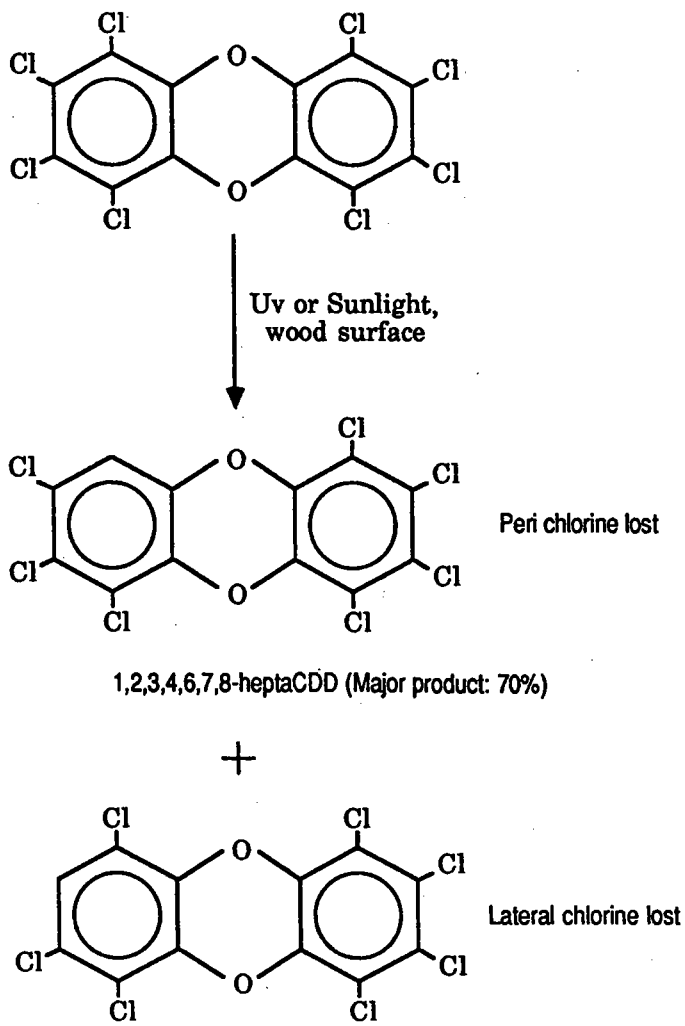
(2,3,7,8-substituted) 1,2,3,7,8-pentaCDD and no 2,3,7,8-tetraCDD (medium not described). Based on these and other studies, the U.S. EPA (1985b) concluded that it is unlikely that photolysis of octaCDD and heptaCDD will produce CDDs chlorinated at all four of the 2,3,7, and 8 positions on the dibenzo-p-dioxin molecule. In other words, breakdown of more highly chlorinated compounds would result in less toxic CDDs.

The U.S. EPA (1985b) conclusion failed to distinguish between laboratory studies and environmental conditions. The fact that irradiation was performed in the presence of hydrogen-donor solutions means that the data can not be directly extrapolated to other environments. Choudhry and Hutzinger (1982) have noted that the type of medium in which CDDs are exposed will affect which chlorines are removed by photolysis. In organic solutions, as noted above by Buser and Rappe (1978), the lateral (2,3,7, and 8) chlorines are removed preferentially. However, on solid media, such as wood surfaces, the peri position (1,4,6 and 9) chlorines are more likely to be removed from octaCDD. This difference was observed when Lamparski et al. (1980) irradiated pentachlorophenol-treated wood under conditions of natural and artificial sunlight. On wood treated with purified pentachlorophenol (very low levels of contaminants), octaCDD was formed by photolytic condensation of pentachlorophenol and subsequently dechlorinated to heptaCDDs and hexaCDDs. The dominant heptaCDD was the 1,2,3,4,6,7,8 isomer (Figure 2.5). Lamparski et al. (1980) stated that "OCDD present on a wood surface is somehow activated so that the preferential chlorine loss occurs at the peri position rather than the lateral position that Buser observed in solution." In this solid phase study, breakdown of higher CDDs resulted in more toxic compounds.

Nestrick et al. (1980) compared photolysis rates for tetraCDD isomers irradiated on a glass surface (solid phase) and in a dilute hydrocarbon solution (solution phase). They examined all 22 tetraCDD isomers and ranked the isomers by relative half-lives. The 2,3,7,8-tetraCDD (all lateral chlorines) had the shortest half-life in a hydrocarbon solution and the longest half-life when exposed as a thin film on a glass surface. In solution phase, the half-life of 2,3,7,8-tetraCDD was 57 minutes; the solid phase half-life was 8,400 minutes. In contrast, the 1,4,6,9 isomer (all peri position chlorines) had the longest half-life in solution. A similar phase-dependent reversal of half-lives was noted for some hexaCDDs and the two heptaCDDs. In hydrocarbon solution, the 1,2,3,6,7,8-hexaCDD had a 50 percent shorter half-life than a mixture of 1,2,3,6,7,9-/1,2,3,6,8,9-hexaCDD. On a glass surface, the half-life of 1,2,3,6,7,8-hexaCDD was more than five times longer than the mixture. Of the two heptaCDDs, 1,2,3,4,6,7,8-heptaCDD had a shorter half-life in

FIGURE 2.5

PHOTOLYTIC DECHLORINATION OF OctaCDD (SOLID PHASE)
(WOOD SURFACE - LOSS OF PERI CHLORINES)
(Data from Lamparski et al., 1980)



solution and a longer half-life on glass than did 1,2,3,4,6,7,9-heptaCDD. It is clear that more studies are necessary to determine specific congeners formed by photolysis of higher CDDs, studies performed both in solution phase and on solid phases such as wood and soil.

Buser (1976) irradiated octaCDD dissolved in a benzene-hexane solution with both gamma rays and UV and compared the dechlorination products. Whereas UV photolysis resulted in preferential loss of lateral chlorines (formation of 1,2,3,4,6,7,9-heptaCDD), gamma irradiation was non-specific and resulted in about equal formation of 1,2,3,4,6,7,8- and 1,2,3,4,6,7,9-heptaCDDs.

CDFs

Less information is available on CDF breakdown reactions. Buser (1976) irradiated octaCDF dissolved in a solution of benzene and n-hexane with both UV and gamma radiation and noted that dechlorination to lower CDFs seemed to be the major reaction pathway. Crosby et al. (1973) reported that 2,8-diCDF dissolved in methanol was rapidly photodegraded to 2-monoCDF under laboratory conditions. Hutzinger et al. (1973) reported the rapid UV photolysis of both 2,8-diCDF and octaCDF in methanol and hexane solutions, resulting in dechlorination with eventual accumulation of "unidentified resinous polymeric-products". The authors noted that the similar photolytic rates in hydrogen-donating solvents for diCDF and octaCDF contrasts with the CDD work of Crosby et al. (1971) where 2,7-diCDD was photodegraded much more rapidly than octaCDD in methanol. In contrast to rapid photolytic rates in hydrogen-donating solvents (20 minutes), transformation rates of thin films of 2,8-diCDF and octaCDF exposed to sunlight in the presence of water were much slower, two months (Figure 2.6; Hutzinger et al., 1973).

Mazer and Hileman (1982) examined the photolysis by UV radiation of 254 nm wavelength in hexane or tetradecane of eight tetraCDFs and derived three guidelines for predicting chlorine loss as tetraCDFs are converted to triCDFS:

1. Chlorines will be removed from the most highly chlorinated ring.

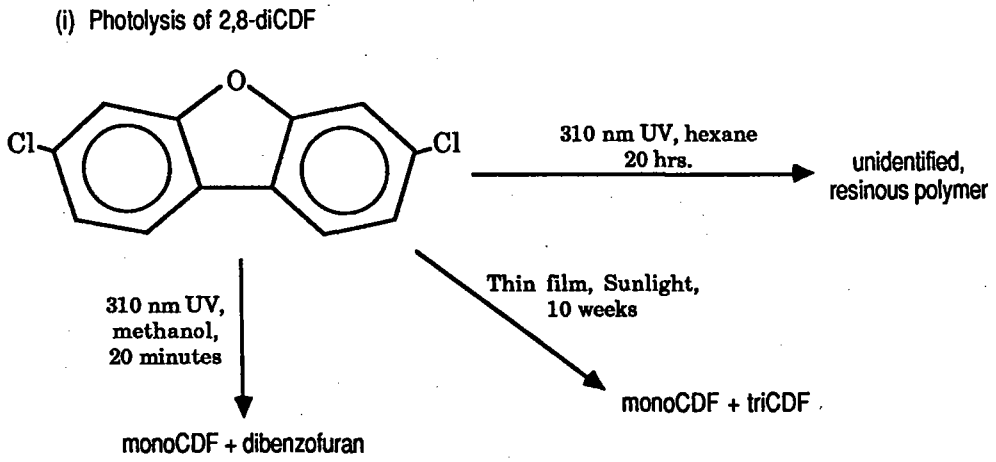
2. The greater the number of adjacent chlorines to a chlorine atom, the greater the likelihood of losing that chlorine.

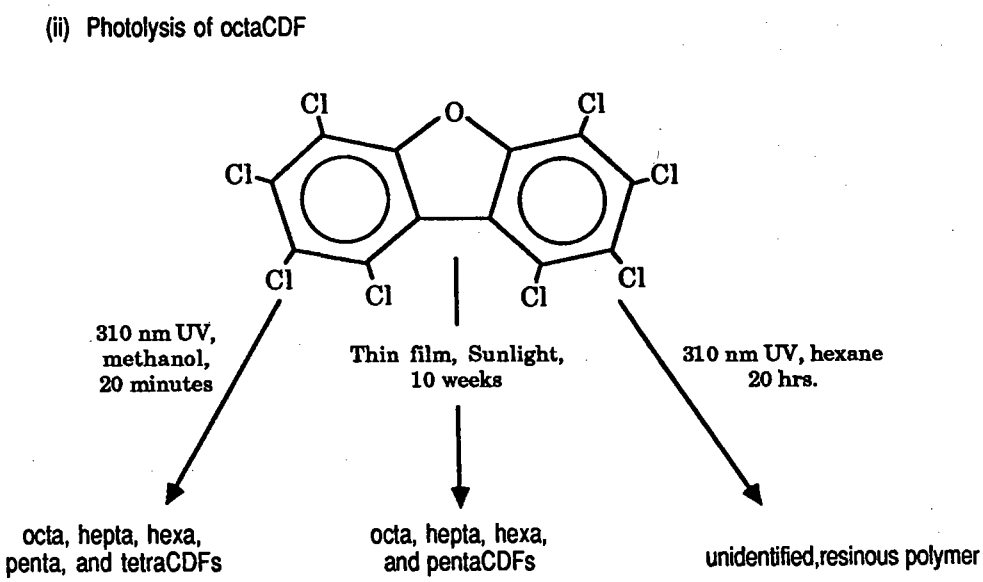
3. Given an equal number of chlorines at the 2 and 3 positions, the 3 position chlorine will be lost first.

FIGURE 2.6

PHOTOLYTIC TRANSFORMATIONS OF PCDFs

(adapted from Huntzinger et al., 1973)





The National Research Council of Canada (1984) discussed the Mazer and Hileman study and noted that, since 254 nm ultraviolet radiation does not reach the earth's surface, the guidelines have "little bearing" on environmental conditions.

MICROBIAL DEGRADATION

Published information on microbial degradation of CDDs is limited (NRCC, 1984). The U.S. EPA (1985a) cited reports that demonstrated lack of microbial degradation of 2,3,7,8-tetraCDD in aquatic systems and by inference concluded that the penta- and hexaCDDs would be even more resistant to microbial breakdown. The biotransformation and biodegradation half-life of 2,3,7,8-tetraCDD in aquatic systems is greater than one year (U.S. EPA 1979a). Some investigators have reported limited microbially mediated degradation of 2,3,7,8-tetraCDD.

Huetter and Philippi (1982) reported very slow microbial degradation of 2,3,7,8-tetraCDD in soil and liquid systems. They found that approximately 1 percent of radiolabelled 2,3,7,8-tetraCDD was converted by single and mixed microbial cultures to an unidentified polar metabolite after several months of incubation. Matsumura and Benezet (1973) examined the ability to degrade 2,3,7,8-tetraCDD of 100 microbial strains known to breakdown resistant pesticides and found only five strains performed any degradation. Quensen and Matsumura (1983) reported oxidative metabolism of 2,3,7,8-tetraCDD present at 5 ppb by two bacterial species and found that metabolism was stimulated by use of ethyl acetate as a carrier solvent. The authors suggested that the solvent may exert a stimulatory effect by aiding 2,3,7,8-tetraCDD penetration of the cell membrane. Thus, restricted microbial uptake could be a limiting factor for environmental degradation of this compound.

Bumpus et al. (1985) recently reported that a common white-rot fungus (Pharnerochaete chrysosporium) is capable of oxidizing 2,3,7,8-tetraCDD to carbon dioxide and attributed this ability to secretion of an extracellular lignin-degrading enzyme system when the fungus was grown in nitrogen-, carbohydrate-, or sulfur-deficient cultures. By showing that this fungus could also convert several other compounds such as DDT and polychlorinated biphenyls to carbon dioxide, the authors demonstrated that this organism is capable of cleaving the ring structure of several halogenated aromatic hydrocarbons. They concluded that, because the degradation is initiated by nitrogen deficiency rather than the level of 2,3,7,8-tetraCDD present, low levels of environmental 2,3,7,8-tetraCDD may provide sufficient substrate for biodegradation.

VOLATILIZATION

Very little information is available on the importance of volatilization for CDF environmental fate, due to lack of water solubility and vapor pressure data (NRCC, 1984). The CARB and CDHS (1986) noted that, as the number of chlorine atoms increases in CDDs and CDFs, the vapor pressures and water solubilities decrease. Thus, the more highly chlorinated the CDD or CDF, the lower expected rate of volatilization (U.S. EPA, 1985a).

The U.S. EPA (1985a) summarized several studies performed on microbial systems and noted that loss by volatilization was considered a major factor in 2,3,7,8-tetraCDD loss. However, there were no quantitative data on rate of volatilization provided in the studies.

Corbet et al. (1983) reported that the highest volatilization rate of radiolabelled 1,3,6,8-tetraCDD from an outdoor pond occurred in the first 24 hours. Marcheterre et al. (1985) reported similar findings for octaCDD.

In a recent review, Mill (1985) stated that volatilization of 2,3,7,8-tetraCDD from soil will be very slow over a wide range of soil moisture content because of low vapor pressure and water solubility. In a study of an herbicide production facility, Thibodeaux (1983) suggested that 29 percent to 46 percent of 2,3,7,8-tetraCDD volatilized from soil over a 350 day period. Muir et al. (1985) studied the fate of radiolabelled 1,3,6,8-tetraCDD in field plots of soil and found that reduction in radioactivity was not due to movement or degradation. They suggested that losses may have occurred by volatilization.

Computer models discussed below have estimated that vaporization will be the major source of loss of 2,3,7,8-tetraCDD from both aqueous and soil systems. In a model predicting 2,3,7,8-tetraCDD loss from aqueous systems, the NRCC (1981) attributed 100 percent of the loss to volatilization, with no appreciable losses to biodegradation or photolysis.

Freeman and Schroy (1985) developed a model to predict the vapor phase migration in soil columns of chemicals possessing low vapor pressures. They applied the model to soil contaminated by 2,3,7,8-tetraCDD in Times Beach, Missouri and made several conclusions about the behavior of this CDD congener:

1. The compound is volatile and its migration through and out of soil is temperature dependent.

2. Because the migration of 2,3,7,8-tetraCDD is highly dependent on depth in the soil column, the environmental fate can not be represented as a simple half-life. For example, the apparent half-life on a soil surface would be on the order of weeks; in contrast, the half-life below a depth of 5 cm would be expressed as years.

3. The compound will volatilize rapidly during summer months, but volatilization will be negligible during the winter.

4. During the first summer after 2,3,7,8-tetraCDD was applied to Times Beach soil, 90 percent of the upper 1 cm layer and 50 percent of the total amount volatilized.

The fate of CDDs and CDFs in the atmosphere is largely unknown because there have been no studies reported on either photochemistry or atmospheric chemistry (CARB and CDHS, 1986). The CARB (1986) noted that these compounds are emitted to the atmosphere in two forms: in the vapor phase and adsorbed to particulates. The fate and persistence will depend in part on the compounds' physical state; the California Air Resources Board (CARB and CDHS, 1986) concluded that CDDs and CDFs will tend to adsorb to particulate matter because of their low vapor pressure.

PERSISTENCE AND MOVEMENT IN SOIL AND SEDIMENTS

CDDs and CDFs are believed to sorb strongly to soils and sediments (U.S. EPA 1985a), and most models predict that soils and sediments will serve as the major sink for these compounds.

Soils

In the absence of organic solvents, leaching and downward migration of CDDs and CDFs to ground water is unlikely (Hutzinger et al., 1985). However, in the presence of organic solvents or in areas possessing sandy soils of low organic content, a greater degree of vertical migration may occur.

CDDs and CDFs in contaminated soil can be spread laterally by wind and soil erosion (Hutzinger et al., 1985). Due to the estimated high affinity of CDFs for soil systems, the NRCC (1984) predicted that these compounds will be highly persistent in the environment. Young (1981) estimated that only about one percent of the 2,3,7,8-tetraCDD remained in an aerial test application site 14 years after spraying. However, Young (1981) noted that, once this compound has bound to soil, its persistence becomes enhanced significantly.

The U.S. EPA (1985a) estimated a soil to water partition coefficient of 48,000 to 1 for soil containing ten percent organic matter. Due to this high affinity for soils, the 2,3,7,8-tetraCDD congener is expected to remain bound at or near the soil surface and apparently becomes more difficult to desorb over time (U.S. EPA 1985a). DiDomenico et al. (1980b) examined 2,3,7,8-tetraCDD levels at 44 contaminated sites at Seveso, Italy: one month after the Seveso chemical plant accident, the 2,3,7,8-tetraCDD half-life in soil was estimated to be on the order of one year. Seventeen months after the incident, the authors estimated the half-life to be greater than ten years. In the previous discussion on volatilization, Freeman and Schroy (1985) concluded that the persistence in soil of 2,3,7,8-tetraCDD could not be expressed as a simple half-life. DiDomenico et al. (1980a) also measured the amount of downward migration at Seveso sites and found 2,3,7,8-tetraCDD at depths of up to 30 cm. However, the highest levels were detected in soil from 0.5 cm to 1.5 cm below the surface.

Freeman and Schroy (1986) modeled the movement of 2,3,7,8-tetraCDD at a site where an herbicide (Agent Orange) contaminated with this compound had been buried. They concluded that vertical migration was very slow, only 10 centimeters over a period of 12 years. Kitunen et al. (1987) examined soil and contamination by chlorinated phenols at four sawmills and reported that while chlorinated phenols were mobile and migrated downward, the CDF (0.2 to 5 ppm) and polychlorinated phenoxyphenol (1 to 50 ppm) contaminants were contained in the top layer of soil.

Although several reviews and models, some cited above, have commented that CDDs are relatively immobile in soils and unlikely to migrate for appreciable distances, recent empirical studies have noted ground water contamination by CDDs and CDFs. Pereira et al. (1985) reported ground water and porous media contaminated by CDDs at depths up to 30 meters at a site in Florida. CDDs and CDFs in both a confined and unconfined aquifer, as well as in soil cores from depths of up to 16 meters, have been detected at Visalia, California (see Chapter 5 of this report).

Recent studies have begun to elucidate these empirical observations of CDD and CDF migration to ground water. Nkedi-Kizza et al. (1985) have noted that at most waste disposal and land treatment sites, soil solutions will consist of both water and mixtures of organic solvents. Most data currently used to predict migration are for sorption of hydrophobic organic compounds from aqueous solution rather than from water-organic solvent mixtures. For sites where organic solvents are present, soil sorption should be characterized by mixtures of water and organic solvent (Nkedi-Kizza et al., 1985).

Jackson et al. (1985) examined the leaching potential of 2,3,7,8-tetraCDD in soils contaminated with chlorinated semi-volatile organic compounds and compared leaching to that in "clean" soils that had been spiked with 2,3,7,8-tetraCDD. There was a strong correlation of leaching potential with solvent-extractable organic content in the soils. They suggested that the presence of halogenated semivolatile compounds as co-contaminants has a major role in regulating 2,3,7,8-tetraCDD solubility and migration in contaminated soil.

Enfield (1985) noted that relatively immobile compounds have been observed to migrate faster than predicted by hydrophic theory. If two percent of total soil fluid is an organic fraction, then hydrophobic theory may underestimate soil mobility of these compounds by a factor of greater than 100. According to Enfield (1985), a partitioning of organic chemicals occurs between water and dissolved organic material. At certain waste disposal sites, both an organic fluid phase and an aqueous phase have been observed to flow through the soil, with the organic phase aiding the transport of hydrophobic chemicals. In his model, Enfield (1985) noted that increased mobility of CDDs is predicted at levels of organic carbon found in the environment (5 to 10 mg/l).

Sediments

Laboratory experiments indicate that 2,3,7,8-tetraCDD is highly sorbed to biological and sediment matrices and more highly chlorinated CDDs are predicted to be concentrated in sediments (U.S. EPA, 1985a). In computer simulated models of an oligotrophic lake and eutrophic pond, the NRCC (1981) determined that the major sinks for 2,3,7,8-tetraCDD would be suspended particles and sediments and that the larger the mass of these sinks, the longer this congener would persist in the environment (NRCC, 1984).

Karickhoff and Morris (1985) discussed the kinetics of sorption of hydrophobic chemicals in sediments and noted that sorption phenomena are frequently described as rapidly reaching equilibrium and as readily reversible. However, sorption under field conditions of highly hydrophobic compounds frequently requires days to weeks in order to reach equilibrium. Karickhoff and Morris (1986) propose a two compartment model, one rapid and readily reversible compartment and a second, slow to reach equilibrium. This approach may be useful to describe the behavior of CDDs and CDFs in sediments.

PLANT UPTAKE

Although removal of CDDs and CDFs from contaminated soils by plant uptake has been proposed as a soil clean-up technique (see Pesticide and Toxic Chemical News, Sept. 18, 1985, p. 12), the data on uptake by terrestial plants are equivocal. The U.S. EPA (1985a) concluded that 2,3,7,8-tetraCDD present in contaminated soil is "not likely" to concentrate in terrestial plants. In contrast to soil systems, the agency (U.S. EPA, 1984a; U.S. EPA, 1985a) cites studies reporting bioaccumulation of this congener by aquatic plants. The National Research Council of Canada's extensive reviews on CDDs (NRCC, 1981) and CDFs (NRCC, 1984) do not address plant uptake. A description of representative terrestrial and aquatic plant studies is provided below.

Terrestial Plants

Several studies have examined vegetable and fruit crop uptake following the accidental 2,3,7,8-tetraCDD release at Seveso, Italy. The Seveso studies have provided contrasting observations. An early study by Coccuci et al. (1979) concluded that plants take up this congener and translocate it to leaves and fruits. In contrast, Wipf et al. (1982) noted that trace levels of 2,3,7,8-tetraCDD were present only on outer surfaces of Seveso fruits and vegetables and attributed the source of this CDD to contaminated dust rather than plant uptake. Recently, in a study using a mixture of contaminated and uncontaminated Seveso soil, Facchetti et al. (1985) reported uptake by vegetables in their root systems. For example, the roots of corn grown in soil with levels at 0.75 ppb 2,3,7,8-tetraCDD contained 1.0 ppb after the roots were rinsed in hexane. However, the authors found only a few ppt in upper portions of vegetables, and they concluded that these low levels of 2,3,7,8-tetraCDD present in above ground parts were transported by volatilization from the soil rather than translocation within the plant.

Young (1981) examined a field sprayed with Agent Orange, an herbicide that contained 2,3,7,8-tetraCDD as a contaminant, and measured 2,3,7,8-tetraCDD levels in roots, stems, and leaves. Young (1981) reported levels in roots and soil were similar at approximately 750 ppt. Levels of 2,3,7,8-tetraCDD in leaves ranged from one to ten percent of root levels. Young did not determine if leaf levels resulted from plant uptake or from contamination by soil particles.

Aquatic Plants

Plant uptake of CDDs in aquatic systems appears to be greater than in terrestial systems. Further, studies are more in agreement for aquatic systems. Tsushimoto et al. (1982) reported rapid accumulation of radioactive 2,3,7,8-tetraCDD by pondweeds. Outdoor ponds were dosed with 54 ppt 2,3,7,8-tetraCDD, and a maximum of 7,000 ppt was concentrated in pondweeds after five days. An equilibrium level of 2,500 ppt in pondweeds was reached after a month. Corbet et al. (1983) reported concentration of radioactive 1,3,6,8-tetraCDD by both floating duckweed and rooted aquatic plants. Maximum levels of the 1,3,6,8-tetra isomer in rooted plants were reached at eight days. Yockim et al. (1978) reported a maximum bioconcentration factor of 2,083 at seven days for 2,3,7,8-tetraCDD by a freshwater alga.

SUMMARY AND DISCUSSION

A summary of environmental fate information for CDDs and CDFs is provided in Table 2.2.

Phototransformation

The CDDs and CDFs can be both formed and broken down by either artificial UV light or sunlight containing UV wavelengths. Under proper conditions, octaCDD can be formed from pentachlorophenol and subsequently dechlorinated to lower chlorinated CDDs. Certain contaminants present in commercial chlorinated phenols can be converted to CDDs and CDFs: predioxins to CDDs and polychlorinated diphenyl ethers to CDFs.

When exposed to ultraviolet (UV) light, CDDs and CDFs will undergo photolysis at a significant rate in the presence of an organic, hydrogen-donating substrate (Crosby et al., 1971). The presence of a hydrogen-donor, which in a number of laboratory experiments has consisted of methanol or hexane, is necessary for a significant amount of photolysis to occur. These conditions may not be met in most environmental situations. The reaction is slow in water and does not occur either on thin layers of pure tetraCDD or on dry soil surfaces (Crosby et al., 1973). The more highly chlorinated CDDs are less reactive than those which are less chlorinated (NRCC, 1981). It has been hypothesized that the low solubility of the more chlorinated CDDs and CDFs in water may retard their photolysis, whereas in an organic hydrogen-donating solvent, the necessary conditions for rapid photolysis are present (Crosby et al., 1981).

TABLE 2.2

SUMMARY OF ENVIRONMENTAL FATE OF CDDs AND CDFs

Process	Medium	Compound or Precursor	Fate or Product	Reference
1. Phototransformation				
a. Formation				
	water,UV	Na-PCP	octaCDD	Crosby et al., 1973
	wood, sunlight	PCP	octaCDD	Lamparski et al., 1980
	methanol, UV	predioxins	CDDs	Nilsson et al., 1974
	hexane, UV	chlorinated diphenyl ethers	CDFs	Norstrom et al., 1976 and 1977
b. Photolysis				
	solution phase: organic solvents, UV or sunlight	higher CDDs and CDFs	lower CDDs and CDFs: preferential removal of lateral (2,3,7,8) chlorines	Buser and Rappe, 1978
	solid phase: wood and glass surfaces, UV or sunlight	higher CDDs	lower CDDs preferential removal of peri (1,4,6,9) chlorines	Lamparski et al., 1980
	benzene-hexane, gamma irradiation	octaCDD octaCDF	lower CDDs and CDFs: non-preferential removal of chlorines	Buser, 1976

TABLE 2.2 (continued)

SUMMARY OF ENVIRONMENTAL FATE OF CDDs AND CDFs

Process	Medium	Compound or Precursor	Fate or Product	Reference
2. Microbial Degradation				
a. fungal catabolism (white rot fungus)	culture medium	2,3,7,8-tetraCDD	carbon dioxide	Bumpus et al., 1985
b. bacterial catabolism	culture medium	2,3,7,8-tetraCDD	unidentified polar metabolite(s)	Huetter and Philippi, 1982; Quensen and Matsumura, 1983
3. Volatilization				
computer simulation	aquatic pond and lake	2,3,7,8-tetraCDD	2,3,7,8-tetraCDD in vapor phase (atmosphere)	NRCC, 1981
4. Persistence and Movement				
a. computer simulation	aquatic pond and lake	2,3,7,8-tetraCDD	bound to sediment and suspended particles	NRCC, 1981
b. migration	waste site containing organic solvents	2,3,7,8-tetraCDD	will migrate with organic fraction, more mobile than predicted in spiked clean soils	Jackson et al., 1985

TABLE 2.2 (continued)

SUMMARY OF ENVIRONMENTAL FATE OF CDDs AND CDFs

Process	Medium	Compound or Precursor	Fate or Product	Reference
5. Plant Uptake				
a. terrestrial plants	soil	2,3,7,8-tetraCDD	uptake by roots	Facchetti et al., 1985
b. aquatic plants and algae	water	2,3,7,8-tetraCDD	uptake with maximum concentration at 5 days	Tsushimoto et al., 1982
		1,2,6,8-tetraCDD	uptake with maximum concentration at 8 days	Corbet et al., 1983

The National Research Council of Canada (NRCC, 1981) has commented that there is little quantitative evidence on rates of CDD photolysis in the natural environment; many studies have been limited to laboratory conditions, using UV wavelengths (less than 290 nanometers) that are screened out by the earth's atmosphere. The U.S. EPA (1985b) stated that photolytic breakdown of CDDs "is not likely to be of environmental importance" in water because these compounds are unlikely to receive UV radiation due to low UV penetration of surface waters and because sorption of CDDs on sediments and suspended particles effectively removes them from solution.

Microbial Degradation

Information on microbial degradation of CDDs and CDFs is very limited (NRCC, 1981; NRCC, 1984). Resistance to microbial attack will increase with increasing chlorination. Overall data support the view that CDDs and CDFs are highly resistant to microbial transformation. A recent report has indicated that a common fungus can break down 2,3,7,8-tetraCDD (Bumpus et al., 1985) under conditions of "nitrogen starvation". The potential role of this fungus in land treatment systems should be examined.

Volatilization

Despite the low vapor pressure of CDDs and CDFs, volatilization is now considered a potential source for loss of these compounds from environmental compartments. In simulating the fate of 2,3,7,8-tetraCDD in two model aquatic ecosystems, the National Research Council of Canada (NRCC, 1981) assigned 100 percent of loss to volatilization and zero to photolysis and microbial degradation. The U.S. EPA (1985b) noted that volatilization is a likely fate for CDDs in aquatic environments. Earlier studies had concluded that volatilization was not an important fate of 2,3,7,8-tetraCDD; for example, in the Water-Related Environmental Fate of 129 Pollutants, the U.S. EPA 1979) stated that volatilization is "probably not an important process" in the aquatic fate of this congener. For CDF volatilization, the NRCC (1984) noted an almost total lack of physical constant data but, by inference from CDD data, predicted low rate constants for volatilization. Nonetheless, the NRCC (1984) concluded that volatilization could play a role in environmental distribution.

Conversion of CDDs and CDFs to the vapor phase does not explain the ultimate fate of these compounds. The California Air Resources Board (CARB and CDHS, 1986) noted that there have been no studies on the behavior of CDDs and CDFs in the vapor phase and there is little knowledge of their fate in the atmosphere, but predicted these compounds would be sorbed to particulate

matter in the air. Summarizing other work, the CARB and CDHS (1986) stated that CDDs and CDFs appear stable when adsorbed to particulate matter, can migrate over great distances in the air, and are probably highly persistent in the atmosphere. Czucwa et al. (1984) found CDDs and CDFs in sediments from a lake located on Isle Royale in Lake Superior and concluded that their presence could only be explained by atmospheric deposition. Thus, while volatilization may remove CDDs and CDFs from aquatic and terrestial compartments, these compounds may be atmospherically transported and subsequently redeposited.

Persistence and Movement in Soil and Sediments

CDDs and CDFs are believed to adsorb strongly to soils, sediments, and biota. Sediments and suspended particulates will serve as sinks for these compounds in aquatic systems (NRCC, 1981); because of strong sorption, they will be highly persistent in the environment. As demonstrated at a site in Visalia, California, these compounds can travel considerable distances downward in soil if organic solvents are present (see Chapter 6: Monitoring). In the absence of organic solvents, CDDs and CDFs are not expected to migrate downward to any great extent. Recent evidence suggests that measurement of CDD movement in soil, based on spiking clean soil with CDDs, does not accurately portray migration at waste disposal and land treatment sites (Nkedi-Kizza et al., 1985; Enfield, 1985; Jackson et al., 1985).

Because these compounds will bind very tightly to organic material in soils and contaminated materials, CDDs and CDFs may escape detection in standard water leachate tests. Use of aqueous leachate tests for these compounds as a screening device prior to land disposal is inappropriate. Rather, a leachate mixture composed of water and organic solvents should be developed and used to determine levels of CDDs and CDFs (Nkedi-Kizza et al., 1985; Jackson et al., 1985). Thus, use of standard leachates required under RCRA for dioxin-containing wastes (U.S. EPA, 1986b) is probably inappropriate because it will underestimate concentrations of CDDs and CDFs in contaminated soils. The treatment standard requires that waste found to contain any tetra-, penta-, or hexaCDD or CDF at levels of 1 ppb or higher in a standard leachate test be treated before land disposal. Young (1981) noted that when soil has been contaminated for several years, the extraction of 2,3,7,8-tetraCDD and subsequent chemical analysis is difficult. The aqueous leachates referenced in the RCRA regulations may not desorb CDDs and CDFs that are highly adsorbed to organic material. In order to extract CDDs and CDFs from soils for chemical analysis, organic solvents are required (see discussion

of chemical analysis in Appendix E). Similar extraction compounds should be used in leachate tests for detection of CDDs and CDFs.

Plant Uptake

There is consensus that plants in aqueous systems take up and concentrate CDDs and CDFs (U.S. EPA 1984a; U.S. EPA 1985b), although Kenaga and Norris (1983) have noted that these compounds may be adsorbed onto external surfaces of aquatic plants rather than actually taken up into plant cells. However, the data for uptake by terrestial plants are less clear. Studies of 2,3,7,8-tetraCDD concentrations in crops grown near the site of the Seveso, Italy chemical accident are contradictory: the presence of this congener in plants is variously attributed to plant uptake and translocation, contaminated dust, and volatilization from soil. After reviewing the literature, the U.S. EPA (1985b) concluded that 2,3,7,8-tetraCDD is "not likely to concentrate in plants grown on contaminated soils."

It should be noted that concentrations of 2,3,7,8-tetraCDD in contaminated soils where plant uptake was examined were on the order of one ppb. Levels of 2,3,7,8-chlorinated CDDs and CDFs encountered at California wood treatment plants and sawmills were much greater (in the high ppb to low ppm range as described in Chapter 5). An important research project would be to determine kinetics of plant uptake where high levels of CDDs and CDFs are present in soils. As Young (1981) has observed, animals foraging on CDD contaminated plants can potentially relocate these compounds off-site.

Land Treatment

In-situ land treatment has been proposed as a potential cleanup method for sites contaminated by CDDs, CDFs, and chlorinated phenols. This option has the potential advantage of being a relatively inexpensive method to clean large volumes of contaminated soil when compared to costs of thermal destruction or removal to distant landfills. Although the inherent assumption is that land treatment will be accomplished by photolysis and perhaps microbial degradation, Young (1981) has observed that reductions in CDD and CDF levels may involve off-site transport, including wind and water movement of contaminated particles, volatilization, and biomass removal. What is needed is a careful study of land treatment, which will examine a number of uncertainties including those listed below.

1. The fate of chlorinated phenols, predioxins, and polychlorinated diphenyl ethers should be examined during land treatment. This is because CDDs and CDFs can be formed from precursor compounds by sunlight under certain conditions. For example, Norstrom et al. (1976) noted the potential in the environment for CDF formation from polychlorinated diphenyl ethers, compounds which are present at up to 100 ppm in commercial pentachlorophenol.

2. Similarly, the dechlorination products from breakdown of higher CDDs and CDFs should be measured. While the U.S. EPA (1985b) noted that the potential for photoformation of 2,3,7,8-tetraCDD from higher chlorinated compounds was unlikely due to preferential removal of the lateral (2,3,7, and 8) chlorine atoms, the U.S. EPA comments appear to be based on a review of solution phase photolysis. In contrast, the findings of Lamparski et al. (1980) indicate that in solid phase photolysis, the potential exists for formation of more toxic CDDs and CDFs by removal of the peri (1,4,6, and 9) chlorine atoms. In land treatment, it should be determined if photodegradation reactions occur as solution phase, solid phase, or a mixture of phases.

3. If a reduction occurs in concentrations of CDDs and CDFs, the ultimate fate of these compounds requires investigation. While Dobbs and Grant (1979) predicted that the most susceptible CDD to photolysis would be 2,3,7,8-tetraCDD, Crosby et al. (1973) reported no 2,3,7,8-tetraCDD degradation in dry soil. Plimmer (1978) suggested that there would be little or no loss of 2,3,7,8-tetraCDD in dry soil from photolysis and that any loss occurring may come from volatilization. Migration by wind blown dust and soil may also account for CDD and CDF removal (Thibodeaux, 1983; Hutzinger et al., 1985).

4. The kinetics of CDD and CDF photolysis during land treatment requires study. Nestrick et al. (1980) noted that some 2,3,7,8-chlorinated CDDs have longer half-lives in solid phase than in organic solution. Solid phase half-lives of 2,3,7,8-tetraCDD and 1,2,3,6,7,8-hexaCDD were over 100 times longer than in organic solution phase. Further, these 2,3,7,8-chlorinated compounds were more resistant to solid phase photolysis than other tetra- and hexaCDDs irradiated.

5. Radiolabelled CDDs and CDFs should be employed during the land treatment study to determine a mass balance for environmental fate of these compounds. That is, the proportion

of these compounds lost by photolysis, microbial breakdown, volatilization, migration on soil particles through wind and water erosion, biological uptake and movement off-site, and atmospheric particulate movement should be identified.

Ideally, a comprehensive land treatment study will explain the fate of these compounds by examining kinetics, formation and degradation products. The use of solvents or other appropriate hydrogen-donating materials should be evaluated. Some work may require specific chemical analysis of 2,3,7,8-chlorinated congeners to insure that increased amounts of 2,3,7,8-chlorinated CDDs and CDFs relative to overall CDD and CDF levels are not formed. In short, if land treatment is a viable disposal technology, it will be so because CDDs and CDFs are destroyed during treatment rather than migrating to other environmental compartments. Because so many questions are unanswered, initial characterization of environmental fate during land treatment should be addressed by laboratory studies prior to full-scale field studies.

CHAPTER 3: AQUATIC TOXICOLOGY

While there are considerable data on the toxicity of some CDDs and CDFs to mammals, aquatic toxicity studies are few and mostly pertain to 2,3,7,8-tetraCDD toxicity.

The first section of this chapter addresses bioconcentration, metabolism and elimination of 2,3,7,8-tetraCDD and other CDDs and CDFs. The second discusses toxicity with the focus mainly on 2,3,7,8-tetraCDD due to lack of information on other CDDs and CDFs.

BIOCONCENTRATION

An organism's uptake and bioconcentration of toxic chemicals depends on factors such as the organism's food intake, surface area to weight ratio, characteristics of the medium in which the organism lives, molecular stability of the chemical, and the organism's metabolism and lipid content (Kenaga and Norris, 1983). The bioconcentration factor (BCF) is a constant proportionality that relates a specific chemical residue in an aquatic organism to the concentration of that chemical in water under standard conditions (Veith et al., 1980). BCFs can be a valuable means of estimating concentrations in water that would pose a threat to aquatic organisms and their consumers. To achieve this, the chemical concentrations in water and tissue must be accurately measured and toxic threshold concentrations in aquatic organisms and their consumers must be known.

Bioconcentration in Fish and Invertebrates

Based on the use of one measured and four estimated octanol/water partition coefficients (K_{ow}), EPA (1984a) predicted BCF values for 2,3,7,8-tetraCDD using six different equations that had been developed by Kenaga and Goring (1980); Veith et al. (1980); and Veith and Kosian (1983). The predicted BCF values derived from the measured partition coefficient (log K_{ow} = 6.15) ranged from 2,870 to 67,800. For the four estimated coefficients (log K_{ow} of 6.84 to 7.28), the predicted BCF values ranged from 6780 to 915,000. However, actual measured BCFs tend to fall at the low end of these predicted values (Table 3.1). The highest measured BCF of 2,3,7,8-tetraCDD reported in the literature for invertebrates is 9,222 in mosquito larvae, Aedes aegypti (Matsumura, 1977). It should be noted that in this study the concentration in water slightly exceeded the water solubility of 2,3,7,8-tetraCDD. The highest average BCF for 2,3,7,8-tetraCDD in fish was 9,270 for rainbow trout (Branson et al., 1985).

TABLE 3.1
2,3,7,8-TETRACDD BIOCONCENTRATION FACTORS FOR AQUATIC ORGANISMS

Species	2,3,7,8-TetraCDD Concentration in Water (ppt)[a]	Bioaccumulation/ Bioconcentration Factor[b]	Exposure Duration[c] (days)	Method	Concentration Initial/Final	Reference
Algae, Oedogonium cardiacum	2.42	2,075	7	Static	Final	Isensee, 1978 (Table 2)
Algae	0.05-239[d]	3,268[e] (avg.)	31	Static	Final	Isensee, 1978 (Table 1)
Pondweeds, Elodea nuttali & cerato-phyllum emersum	53.7[f]	130 (max. conc.) (at 5 days)	5->60	Static (pond water)	Initial	Tsushimoto et al., 1982
Brine shrimp (Artemia salina)	100	1,570	4-7	Static	Final	Matsumura, 1977
Snail, Physa sp.	2.42	2,095	7	Static	Final	Isensee, 1978 (Table 2)
Snail	0.05-239[d]	6,106 (avg.)[e]	31	Static	Final	Isensee, 1978 (Table 1)
Mosquito larvae; (Aedes egypti)	450[g]	9,222	4-7	Static	Final	Matsumura, 1977
Daphnia magna	2.42	7,070	7	Static		Isensee, 1978 (Table 2)
	0.05-239[d]	4,438 (avg.)[e]	31	Static	Final	Isensee, 1978 (Table 1)
Channel catfish Ictalurus punctatus	0.05-239[d]	2,203 (avg.)	31	Static	Final	Isensee, 1978 (Table 1)
Mosquitofish Gambusia affinis	2.4	4,875	7	Static	Final	Yockim et al., 1978
	0.05-239[d]	6,970 (avg.)[e]	3	Static	Final	Isensee, 1978 (Table 1)
	2.42	4,850	7	Static	Final	Isensee, 1978 (Table 2)
Rainbow Trout	107	9270	6 hours	Static	Final	Branson et al., 1985

a/ All concentrations were analytically determined except Tsushimoto et al., 1982.

b/ Based on ^{14}C count as ^{14}C tetraCDD whole body, average values, wet weight.

c/ Soil treated with 2,3,7,8-tetraCDD and added to an aquatic ecosystem except for Tsushimoto et al., 1982 where 2,3,7,8-tetraCDD was added directly to pond water.

d/ Bioaccumulation Ratios (BR) were averages of several experiments using concentrations ranging from .05 - .239 ng/l. One of the concentrations (1330 ng/l) was unacceptably greater than the solubility of 2,3,7,8-tetraCDD (200 ng/l). Its BR was not included in the averages.

e/ Bioaccumulation ratios-organisms were exposed with other organisms.

f/ Estimated value assuming a homogenous distribution in water.

g/ This water concentration is slightly greater than water solubility of 2,3,7,8-tetraCDD (200 ppt).

Much attention has been given to studying 2,3,7,8-tetraCDD because of its known mammalian toxicity. Kuehl et al. (1985b) examined the uptake and bioconcentration of all 22 possible tetraCDD isomers in carp fry during 30 day exposures to two types of incinerator fly ash, "east coast" and "midwest" (Table 3.2 provides fly ash composition.) Although other congeners were present in the fly ashes, only the tetraCDDs were looked for. Municipal incinerator fly ash is thought to be a source of 2,3,7,8-tetraCDD contamination in the Great Lakes watershed. In this "soup" type of exposure to mixtures of CDDs and CDFs, preferential accumulation and retention of 2,3,7,8-tetraCDD occurred. No other tetraCDDs were detected. This finding was observed in both static and flow-through tests and for 2,3,7,8-tetraCDD bound either to fly ash or released from the fly ash extract. The 2,3,7,8-tetraCDD was concentrated 100 times more from the extract than from the solid matrix. East Coast fly ash contained higher levels of 2,3,7,8-tetraCDD and four times more organic carbon than the midwest fly ash. However, carp concentrated 2,3,7,8-tetraCDD from the midwest fly ash to a greater extent than the east coast fly ash. The authors suggest that the availability of 2,3,7,8-tetraCDD may be inversely related to the amount of organic carbon present in the fly ash.

In a subsequent study that included additional CDD and CDF isomer groups, Kuehl et al. (1986a) exposed carp to fly ash for 60 days. The authors found that carp bioconcentrated not only 2,3,7,8-tetraCDD but also penta-, hexa-, and hepta- congeners of CDDs and CDFs chlorinated in the 2,3,7, and 8 positions (Table 3.3). In a related study, Kuehl et al. (1986b) exposed carp to sediment from a Wisconsin reservoir containing several CDDs and CDFs (Tables 3.4 and 3.5). A pattern of selective accumulation of 2,3,7,8-chlorinated congeners was observed, with the highest accumulation by 2,3,7,8-tetraCDD. Accumulation of only two non-2,3,7,8-chlorinated compounds from sediment, a pentaCDF and a hexaCDF, was detected.

Muir et al. (1985b) observed the bioconcentration of ^{14}C labelled 1,2,3,7-tetraCDD, 1,2,3,4,7-pentaCDD, 1,2,3,4,7,8-hexaCDD, and 1,2,3,4,6,7,8-heptaCDD in juvenile fathead minnows and rainbow trout by exposure to each of these congeners separately over a five day period. In a second study with a similiar exposure regime, Muir et al. (1986) exposed these same species to ^{14}C labelled 1,3,6,8-tetraCDD and octaCDD. The highest BCFs for isomers in both these studies averaged 4,232 for 1,2,3,4,7,8-hexaCDD and 5,702 for 1,3,6,8-tetraCDD for fathead minnows (Table 3.6). These BCFs are approximately one half the BCFs reported for 2,3,7,8-tetraCDD of 9,270 for rainbow trout (Branson et al., 1985).

Corbet et al. (1983) exposed fathead minnows and rainbow trout for 96 hours to 20 ng/l carbon 14-labelled 1,3,6,8-tetraCDD. They reported steady state BCF's of 610 and 210, respectively.

TABLE 3.2

CDD CONCENTRATIONS IN EAST COAST AND MIDWEST FLY ASH
(Adapted from Kuehl et al., 1985b)

CDD	Midwest Fly Ash pg/g [a/]	East Coast Fly Ash pg/g [b/]
Tetrachloro-*		
2378	160	2,000
1469	55	600
1269	180	2,400
1267	86	1,500
1289	72	1,600
1369	970	5,200
1247 + 1248	2,200	19,000
1278	860	6,800
1268	1,000	8,900
1237 + 1238	3,600	26,000
1279	280	5,500
1246 + 1249	210	3,500
1478	180	2,300
1236	290	3,500
1239	250	3,700
1246 + 1249	210	2,100
1368	17,000	48,000
1379	13,000	45,000
1378	2,200	21,000
1234	550	16,000
Total	43,353	224,900
Hexachloro-**		
124679 + 124689	8,800	15,000
123468	46,000	35,000
123679 + 123689	30,000	32,000
123469	-	3,700
123478 + 123678	11,000	15,000
123467 + 123789	8,000	14,800
Total	103,800	165,600

TABLE 3.2 (continued)

CDD CONCENTRATIONS IN EAST COAST AND MIDWEST FLY ASH
(Adapted from Kuehl et al., 1985b)

CDD	Midwest Fly Ash pg/g [a/]	East Coast Fly Ash pg/g [b/]
Heptachloro-**		
1234679	36,000	54,000
1234678	42,000	53,000
Total	78,000	107,000
Octachloro-**		
12346789	52,000	95,000
Organic Carbon	1%	4%

* corrected for percent recovery of $^{13}C_{12}$ 2,3,7,8-tetraCDD
** absolute values not corrected for recovery
a/ from a midwestern municipal incinerator
b/ a blend of fly ash from 5 different municipal incinerators from the east coast.

TABLE 3.3

CONCENTRATIONS OF CDDs AND CDFs IN CARP TISSUE AFTER 60 DAY EXPOSURE TO FLY ASH CONTAINING VARIOUS CDDs AND CDFs (Adapted from Kuehl, 1985a)

		Fly Ash[a/] (pg/g)	Carp (pg/g)
	1,3,6,8-tetraCDD	48,000	ND [b/]
	1,3,7,9-tetraCDD	45,000	ND
*	2,3,7,8-tetraCDD	2,000	7.5
*	1,2,3,7,8-pentaCDD	-	43
*	1,2,3,6,7,8/1,2,3,4,7,8-hexaCDD	15,800	105
*	1,2,3,4,6,7,8-heptaCDD	53,000	104
	1,2,3,4,6,7,9-heptaCDD	54,000	2
*	2,3,7,8-tetraCDF	-	7.2
*	1,2,3,7,8-pentaCDF	-	14
*	1,2,3,6,7,8-hexaCDF	-	24
*	1,2,3,4,6,7,8-heptaCDF	-	27

a/ A blend of flyash from 5 different incinerators on the east coast with 4 percent organic carbon. Dashes signify that no chemical analyses were performed

b/ ND = not detected

* Congeners chlorinated in the 2,3,7, and 8 positions

TABLE 3.4

RESULTS OF CDD ANALYSIS OF WISCONSIN RESERVOIR SEDIMENT AND FISH
(Adapted from Kuehl et al., 1986b)

CONGENER	CONCENTRATIONS	
	Sediment (pg/g)	Carp (pg/g)
TetraCDD		
1,3,5,8-	17	ND**
*2,3,7,8-	170	120
PentaCDD		
1,2,4,6,8-; 1,2,4,7,9-	136	ND
1,2,3,6,8-	53	ND
1,2,4,7,8-	36	ND
1,2,3,7,9-	15	ND
1,2,3,4,7-; 1,2,4,6,9-	53	ND
*1,2,3,7,8-	31	4.8
1,2,3,6,9-	14	ND
1,2,4,6,7-; 1,2,4,8,9-	23	ND
1,2,3,6,7-	11	ND
1,2,3,8,9-	5	ND
HexaCDD		
1,2,4,7,9-; 1,2,4,6,8,9-; 1,2,3,4,6,8-	1090	ND
1,2,3,6,7,9-; 1,2,3,6,8,9-	580	ND
*1,2,3,6,7,8-	180	16
1,2,3,4,6,9-	16	ND
*1,2,3,7,8,9-	60	ND
HeptaCDD		
*1,2,3,4,6,7,8-	2190	27
1,2,3,4,6,7,9-	4720	ND
OctaCDD		
*1,2,3,4,6,7,8,9-	20,560	25

* Chlorinated at the 2,3,7, and 8 positions
** ND not detected; minimum level of detection, 1 pg/g

TABLE 3.5

RESULTS OF CDF ANALYSIS OF WISCONSIN RESERVOIR SEDIMENT AND FISH
(Adapted from Kuehl et al., 1986b)

CONGENER	CONCENTRATIONS	
	Sediment (pg/g)	Carp (pg/g)
TetraCDF		
1,3,7,8-	6	ND **
1,3,4,6-; 1,2,4,8-	20	ND
1,2,4,6-	14	ND
1,2,3,7-; 1,2,6,8-: 1,4,7,8-: 1,3,6,9-	15	ND
1,2,3,8-; 1,4,6,7-: 2,4,6,8-: 1,2,3,6-	31	ND
1,2,7,8-	88	ND
1,2,6,7-; 1,2,7,9-	10	ND
1,2,4,9-; 2,3,6,8-	19	ND
2,4,6,7-	7	ND
*2,3,7,8-	182	28
2,3,6,7-	24	ND
3,4,6,7-	5	ND
1,2,8,9-	8	ND
PentaCDF		
1,2,4,6,8-	64	ND
1,2,3,6,8-; 1,3,4,7,9-	9	ND
1,2,4,7,8-	22	ND
1,2,4,7,9-; 1,3,4,6,7-	3	ND
1,2,4,6,7-	8	ND
1,2,3,4,7-; 2,3,4,6,9-	4	ND
*1,2,3,4,8-; 1,2,3,7,8-	14	2.6
2,3,4,6,8-; 1,2,4,6,9-	9	ND
2,3,4,8,9-	6	ND
1,2,4,8,9-	5	ND
*2,3,4,7,8-	8	4.4
1,2,3,8,9-	2	ND
2,3,4,6,7-	2	2.8

TABLE 3.5 (continued)

RESULTS OF CDF ANALYSIS OF WISCONSIN RESERVOIR SEDIMENT AND FISH
(Adapted from Kuehl et al., 1986b)

CONGENER	CONCENTRATIONS	
	Sediment (pg/g)	Carp (pg/g)
HexaCDF		
1,2,3,4,6,8-	21	ND
1,3,4,6,7,8-	91	ND
1,2,3,4,7,9-; 1,2,3,4,7,8-	30	ND
*1,2,3,6,7,8-	11	1
1,2,3,4,6,7-	84	2
1,2,3,6,8,9-; 1,2,3,4,8,9-	6	ND
HeptaCDF		
*1,2,3,4,6,7,8-	290	2.5
1,2,3,4,6,8,9-	430	ND
OctaCDF		
1,2,3,4,6,7,8,9-	850	ND(5)***

* Chlorinated at the 2, 3, 7, and 8 positions
** ND not detected, minimum level of detection, 1 pg/g
*** isomer detected but analysis did not meet quality assurance criteria at 5 pg/g

TABLE 3.6

BIOCONCENTRATION OF CDD COMPOUNDS OTHER THAN 2,3,7,8-TETRACDD IN RAINBOW TROUT FRY AND FATHEAD MINNOWS (Adapted from Muir et al., 1985b and 1986)

Compound	Experiment Number	Conc.[a] in Water ng/l	BCF[b]
Rainbow Trout			
1,3,6,8-tetraCDD	1	4	2938 ± 480
	2	74	1964 ± 223
	3	211	1400 ± 473
1,2,3,7-tetraCDD	1	134	874 ± 83
	2	54	1577 ± 24
1,2,3,4,7-pentaCDD	1	16	810 ± 20
1,2,3,4,7,8-hexaCDD	1	47	1715 ± 112
	2	10	2840 ± 331
1,2,3,4,6,7,8-heptaCDD	1	55	1059 ± 91
	2	11	1790 ± 353
OctaCDD	1	415	34 + 18
	2	20	136 ± 55
Fathead Minnows			
1,3,6,8-tetraCDD	1	10	5840 ± 2859
	2	41	5565 ± 1550
1,2,3,7-tetraCDD	1	23	2018 ± 1
	2	28	2458 ± 206
1,2,3,4,7-pentaCDD	1	19	1647 ± 361
	2	11	1220 ± 157
1,2,3,4,7,8-hexaCDD	1	18	2630 ± 130
	2	7	5834 ± 1038
1,2,3,4,6,7,8-heptaCDD	1	39	513 ± 46
	2	8	515 ± 167
OctaCDD	1	9	2226 ± 1067

a/ Time-weighted average centrifuged water concentration during uptake.

b/ Bioconcentration factors are plus or minus the standard deviation.

Thus, it appears that non-2,3,7,8-chlorinated congeners can be bioconcentrated in aquatic organisms.

Two studies have elucidated target tissues for 2,3,7,8-tetraCDD uptake and retention. Kuehl et al. (1986b) examined the distribution of 2,3,7,8-tetraCDD in several specific tissues of male and female carp taken from a contaminated Wisconsin reservoir. Fillet, liver, visceral fat, brain, and cranial fat were analyzed. There appeared to be a greater deposit of 2,3,7,8-tetraCDD in fatty tissues, especially in cranial fat (Table 3.7).

TABLE 3.7

ORGAN-SPECIFIC 2,3,7,8-TETRACDD ANALYSIS OF MALE AND FEMALE CARP
(Adapted from Kuehl et al., 1986b)

ORGAN	MALE (n=7) (pg/g)*	FEMALE (n=5) (pg/g)*
Fillet	23	28
Liver	93	150
Visceral Fat	280	300
Brain	68	24
Cranial Fat	370	370

*Detection limit: 1 pg/g.

The distribution of radiolabelled 2,3,7,8-tetraCDD fed 494 ng/kg/day to juvenile rainbow trout and yellow perch was determined after 13 weeks exposure (Kleeman et al., 1986a, 1986b). Rainbow trout had high concentrations in visceral fat, pyloric caeca, and the carcass; the carcass had the highest lipid concentration (Table 3.8). In yellow perch the visceral fat and liver had high concentrations of 2,3,7,8-tetraCDD but lipid concentrations for these were not given (Table 3.9).

Several of the bioconcentration studies reviewed have used contaminated sediment as the method of exposure. In these types of tests the BCF usually is based on the compound's concentration in water after its desorption from sediment. This experimental design was thought to prevent the compound's concentration in water from exceeding its solubility. However, for highly

TABLE 3.8

2,3,7,8-TETRACDD
CONCENTRATION AND LIPID CONCENTRATION
IN ORGANS OF RAINBOW TROUT[a/]
(Adapted from Kleeman et al., 1986a)

ORGAN	2,3,7,8-TETRACDD[b/] CONCENTRATION	LIPID CONCENTRATION (g lipid/g tissue)
Visceral fat	3269 ± 667	0.98 ± 0.01
Pyloric caeca	355 ± 79	0.48 ± 0.06
Carcass	315 ± 25	0.55 ± 0.02
Gill	244 ± 19	
Skin	201 ± 40	0.29 ± 0.01
Gastrointestinal tract	102 ± 14	
Kidney	92 ± 11	
Spleen	85 ± 10	
Liver	72 ± 9	
Heart	70 ± 3	
Skeletal Muscle	29 ± 3	0.14 ± 0.01

a/ Trout were killed at week 13 for determination of 2,3,7,8-tetraCDD concentration and at week 14 for lipid concentration. Values are mean ± SE of four to six fish.

b/ pg equivalent concentration of ^{3}H tetraCDD/g

TABLE 3.9

2,3,7,8-TETRACDD
CONCENTRATION AND LIPID CONCENTRATION
IN ORGANS OF YELLOW PERCH[a/]
(Adapted from Kleeman et al., 1986b)

ORGAN	2,3,7,8-TETRACDD[b/] CONCENTRATION	LIPID CONCENTRATION (g lipid/g tissue)
Visceral fat	2769 ± 134	
Liver	466 ± 33	
Spleen	166 ± 57	
Gill	155 ± 16	
Gastronintestinal tract	148 ± 19	
Pyloric caeca	143 ± 6	
Carcass	129 ± 7	0.19 ± 0.03
Kidney	119 ± 28	
Heart	77 ± 9	
Skin	41 ± 5	0.03[c/]
Skeletal muscle	9 ± 1	0.05 ± 0.01

a/ Yellow perch were killed at week 13 for analysis of 2,3,7,8-tetraCDD and lipid concentrations. Values are mean ± SE of six fish.

b/ pg equivalent concentration of ^{3}H tetraCDD/g

c/ Analysis of lipid concentration of skin pooled from six fish.

lipophilic compounds, the presence of dissolved organic carbon in water can sometimes allow a compound's concentration to exceed its water solubility.

Muir et al. (1984) designed an experiment to assess the influence of chemical properties, sediment type, and species characteristics on the bioavailability of CDDs to aquatic insects. They exposed five species of burrowing and nonburrowing insects to sand and sandy silt sediments containing either radiolabelled 1,3,6,8-tetraCDD or octaCDD. Test animals were exposed either directly to sediments or indirectly in waters overlying sediments. BCFs were determined based on 96-hour exposure (Table 3.10).

Two observations were made concerning the chemical characteristics of these two congeners (1,3,6,8-tetraCDD and octaCDD) with respect to bioavailability. First, the test animals had much lower BCFs for 1,3,6,8-tetraCDD and octaCDD than predicted from their water solubilities and partition coefficients. The authors suggest, that for octaCDD, the lower BCF may be due to its extreme hydrophobicity resulting in strong adsorption to sediment and dissolved organic carbon and to poor absorption across biological membranes due to steric factors. Alternatively, low BCFs could result from overestimation of the concentration in test water due to possible association with dissolved organic carbon, a form less suitable for bioavailability. In this study, octaCDD was present in concentrations which, after centrifugation, greatly exceeded its solubility. This phenomenon of octaCDD concentration apparently exceeding its water solubility has also been noted in other studies with octaCDD (Bruggeman et al., 1984; and Muir et al., 1986). The authors suggest that overestimation could occur because the measured radioactivity in water could include degradation products; these are possibly more polar and therefore less efficiently accumulated by the insects. Analytical confirmation for specific congeners was not conducted.

The second observation was that 1,3,6,8-tetraCDD was concentrated to a higher degree than octaCDD by all non-burrowing and burrowing insects. One reason may be that the structure of 1,3,6,8-tetraCDD is similar to 2,3,7,8-tetraCDD, which has better uptake than other congeners due to its optimal steric configuration. In bioaccumulation studies of superlipophilic chemicals, Bruggeman et al. (1984) found negligible accumulation of octaCDD in fish from aqueous and oral exposures. The author suggested that uptake is hindered by particular structural or physico-chemical properties interfering with membrane transport, such as molecular mass or size.

TABLE 3.10

BIOCONCENTRATION FACTORS FOR BURROWING AND NON-BURROWING INSECTS EXPOSED TO CDD CONTAMINATED SEDIMENT
(Adapted from Muir et al., 1984)

Animal	Sediment Type	Water Concentration (ng/l)[1/] % Sorbed (in parentheses) (96 hr)	Conc. in Pore Water[3/] (96 hr)	96 hr BCF in Water or Sediment[2/] BCFw	BCFsw	BCFs
1,3,6,8-TCDD						
Chironomus[4/]	Sand	366 (45)	3,843	1,554 ± 249	4,135 ± 185 *	394 ± 18
	Silt	44 (80)	57	1,992 ± 92	4,682 ± 1,042 *	3,602 ± 802 *
Hexagenia[4/]	Sand	166 (70)	1,743	849 ± 254	5,291 ± 619 *	504 ± 59
	Silt	55 (96)	72	2,846 ± 983	5,399 ± 1,506 *	4,153 ± 1,158 *
Paragnetina[5/]	Sand	205 (58)	2,152	830 ± 69	1,048 ± 364	100 ± 35
	Silt	142 (34)	185	———	136 ± 20	105 ± 15
Acroneuria[5/]	Sand	" "	"	794 ± 211	963 ± 95 **	92 ± 9
	Silt	" "	"	———	182 ± 77	140 ± 59
Pteronarcys[5/]	Sand	" "	"	———	843 ± 157	80 ± 15
OCDD						
Chironomus[4/]	Sand	235 (71)	1,814	141 ± 104	183 ± 70	24 ± 9
	Silt	101 (81)	101	145 ± 95	77 ± 44	77 ± 44
Hexagenia[4/]	Sand	102 (67)	787	———	1,086 ± 465	141 ± 60
	Silt	24 (99)	24	———	1,019 ± 427	1,109 ± 427
Paragnetina[5/]	Sand	182 (36)	1,405	42 ± 13	81 ± 19 *	11 ± 2
	Silt	147 (41)	147	———	15 ± 2	15 ± 2
Acroneuria[5/]	Sand	" "	"	———	99 ± 38	13 ± 5
	Silt	" "	"	———	20 ± 7	20 ± 7
Pteronarcys[5/]	Sand	" "	"	160 ± 6	156 ± 45	20 ± 6

1/ = Water concentration after centrifugation at 20,000 g (30 min). Mean of duplicate samples.
2/ = BCFw = average concentration insects in water (96 hr)/average water conc. over interval;
BCFsw = average concentration insects in sediment (96 hr)/average water conc. and
BCFs = average concentration insects in sediment (96 hr)/pore water concentration.
3/ = Pore water concentration was determined on the supernatant of centrifuged wet sediment.
4/ = Chironomus and Hexagenia are burrowing insects (detritivores).
5/ = All water data for stonefly nymphs (Paragnetina, Acroneuria, and Pteronarcys) are combined (Means of 6 samples). These are non-burrowing insects.
* = Indicates significant difference between BCFw and BCFs or BCFsw at P = 0.01.
** = Indicates significant differences between BCFw and BCFs or BCFsw at P = 0.05.

Species characteristics and sediment type affected the bioaccumulation of 1,3,6,8-tetraCDD (Muir et al., 1984). The burrowing insects (detritivores) had higher BCFs for 1,3,6,8-tetraCDD than the non-burrowing insects for all types of exposures. BCFs were especially high for those detritivores exposed to a sediment containing silt. Ingestion of sediment during the burrowing activity of the detritivores, especially mixtures of sand and silt that contain a size range of particles favored by these animals, may explain their greater BCFs. These organisms also had come into contact with pore water (water within passageways in the sediment), which was generally more contaminated than the water above the sediment. This difference in BCFs between the burrowing insects and non-burrowing insects was not observed in any of the octaCDD exposures.

Isensee (1978) and Isensee and Jones (1975) exposed daphnids, mosquitofish, catfish, and snails to 2,3,7,8-tetraCDD-contaminated sediment and measured concentrations in sediment, tissue, and water. The bioconcentration factors averaged over a range of test concentrations for these organisms were 4,438, 6,970, 2,203, and 6,106, respectively. They found significant correlations ($r = 0.94$ to .97) between 2,3,7,8-tetraCDD concentrations in water and tissues for a wide range of water concentrations. The authors concluded that the amount of 2,3,7,8-tetraCDD accumulated by the organisms in this test was controlled almost entirely by the amount of 2,3,7,8-tetraCDD available in the water. However, correlations for sediment and tissue were not calculated and one might argue that catfish and snails ingest a significant amount of sedimentary material while feeding.

Kuehl et al. (1986b), as discussed above, reported levels of CDDs and CDFs in carp exposed to contaminated sediment. These fish, although not inhabitants of bottom mud or soil, were found in this study to contain large amounts of sediment in their intestines. The authors, in an attempt to determine whether these carp (exposed in the laboratory) had accumulated 2,3,7,8-tetraCDD from water across the gills, analyzed the bioassay water after centrifugation and did not find 2,3,7,8-tetraCDD. They contended that the rate of CDD desorption from sediment to water while passing over the gills is too slow to influence the amount of CDD available for uptake by that route. They concluded that the level of 2,3,7,8-tetraCDD detected was essentially from sediment that passed through the gut during the course of normal feeding behavior.

Bioconcentration In Aquatic Plants

Three studies have investigated bioconcentration of 2,3,7,8-tetraCDD by aquatic plants. Isensee and Jones (1975); and Isensee (1978) reported average bioaccumulation ratios of 3,260 and 2,075 for an alga, Oediogonium cardiacum after a 31 day exposure. Pondweeds, Elodea nuttali and Ceratophyllon demersum, reached a maximum bioconcentration factor of 130 after 5 days of a 50 day study in a manmade outdoor pond (Tsushimoto et al., 1982). Kenaga and Norris (1983) criticized reported bioconcen- values as being strongly affected by adsorption of 2,3,7,8-tetraCDD onto the surface of plants. The methods used in these studies to determine 2,3,7,8-tetraCDD concentrations did not consider adsorption.

Metabolism

To study the metabolism of 2,3,7,8-tetraCDD in fish, Kleeman et al. (1986a and 1986b) analyzed the liver, kidney, and skeletal muscle of adult yellow perch and rainbow trout one week after a single injection of 60 ug/kg radiolabelled 2,3,7,8-tetraCDD. In these tissues, the parent compound accounted for all of the extractable ^{14}C in rainbow trout and 96 to 99 percent in yellow perch. However, the gallbladder bile of both of these species contained the parent compound and 2,3,7,8-tetraCDD metabolites. At least one of the metabolites in the bile of both species was a glucuronide conjugate.

Depuration and Elimination

Water Exposure

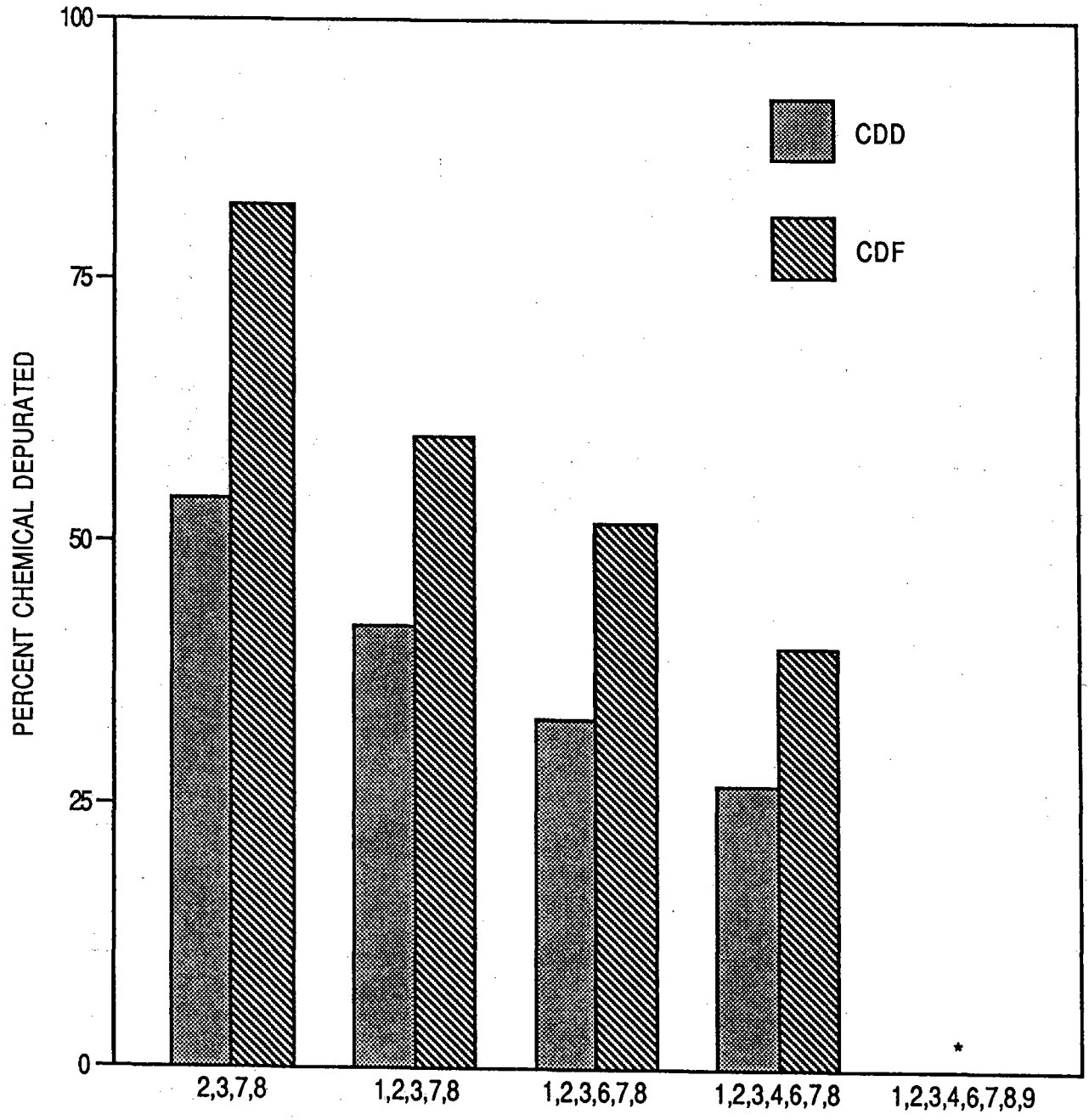
Kuehl et al. (1985a, 1986b) attempted to determine the accumulation and depuration of 2,3,7,8-tetraCDD in carp exposed to sediment containing 39 pg/g 2,3,7,8-tetraCDD. After 55 days of exposure, the carp (5 to 8 percent lipid) had accumulated 2,3,7,8-tetraCDD to a level of 7.5 pg/g; however, a steady state had not yet been reached. The fish were then placed in clean water to observe depuration. At 205 days only 33 percent depuration had occurred.

In the same study, carp (15 to 18 percent lipid) were taken from a contaminated reservoir in Wisconsin and maintained in clean water for 336 days. The carp lipid fraction was analyzed for 2,3,7,8-tetraCDD on days 1, 64, 119, 224, and 336. Other 2,3,7,8-chlorinated CDDs and CDFs were also analyzed for on days 1 and 336 (Figure 3.1 and Table 3.11). Depuration rates of both CDDs and CDFs decreased with increasing chlorination. For the same degree of chlorination, the percentage of CDFs depurated

Figure 3.1

DEPURATION OF 2,3,7,8-SUBSTITUTED CDDs AND CDFs FROM CARP AFTER 336 DAYS

(Adapted from Kuehl et al., 1985b)



* NO CHANGE IN CONCENTRATION DETECTED.

TABLE 3.11

DEPURATION OF 2,3,7,8-SUBSTITUTED CDDs AND CDFs FROM CARP AFTER 336 DAYS[a/]
(Data from Kuehl et al., 1985a)

Compound	Day 1	Day 336	Percent Depurated
CDD			
2,3,7,8-tetraCDD	370	170	54
1,2,3,7,8-pentaCDD	13	7.5	42
1,2,3,6,7,8-hexaCDD	24	16	33
1,2,3,4,6,7,8-heptaCDD	30	22	27
1,2,3,4,6,7,8,9-octaCDD	38	38	0
CDF			
2,3,7,8-tetraCDF	150	27	82
1,2,3,7,8-pentaCDF	4.9	1.9	60
1,2,3,6,7,8-hexaCDF	5.4	2.6	52
1,2,3,4,6,7,8-heptaCDF	5.3	3.2	40
1,2,3,4,6,7,8,9-octaCDF	12	12	0

a/ Expressed as pg/g lipid.

---

was greater than that for CDDs. No depuration of octaCDD or octaCDF was observed. The half-life of 2,3,7,8-tetraCDD in tissues was found to be approximately 300 to 320 days (Kuehl et al., 1985a, 1986b). Half-lives for CDF isomers in this study were not reported but tetra-, penta-, and hexaCDFs had depurated over 50 percent by day 336. Hepta- and octaCDFs had not reached 50 percent depuration.

The shortest depuration half-life reported for 2,3,7,8-tetraCDD is 58 days for rainbow trout after a six hour exposure to 107 ng/l 2,3,7,8-tetraCDD in water (Branson et al., 1985). This half-life is still far greater than any other isomers studied.

Muir et al. (1985b and 1986) observed the depuration half-lives in rainbow trout and fathead minnows exposed through water for five days to concentrations of six congeners from five different isomer groups (Table 3.12). The half-lives for the hexa- and

TABLE 3.12

HALF-LIVES OF CDD COMPOUNDS OTHER THAN 2,3,7,8-TETRACDD IN FISH
(Adapted from Muir et al., 1985b and 1986)

SPECIES AND CONGENER	AVERAGE HALFLIFE (days)
Rainbow Trout	
1,2,6,8-tetraCDD	7.5
1,2,3,7-tetraCDD	2.7
1,2,3,4,7-pentaCDD	2.5
1,2,3,4,7,8-hexaCDD	15
1,2,3,4,6,7,8-heptaCDD	16.7
OctaCDD	6
Fathead Minnow	
1,3,6,8-tetraCDD	7.5
1,2,3,7-tetraCDD	2.8
1,2,3,4,7-pentaCDD	3.2
1,2,3,4,6,7,8-hexaCDD	23.5
1,2,3,4,6,7,8-heptaCDD	17.5
OctaCDD	13

heptaCDDs were longer than for the lower chlorinated tetra- and pentaCDDs, which corresponds to higher octanol/water partition coefficients for more highly chlorinated CDDs. However, the elimination half-life of octaCDD in rainbow trout was almost equal to 1,3,6,8-tetraCDD. A decrease in the depuration of CDDs from carp as the degree of chlorination increased was observed by Kuehl et al. (1985a, 1986b) for 2,3,7, and 8 chlorinated congeners including octaCDD. The water concentration of octaCDD was much greater than its solubility. As a result, the authors suspect that the octaCDD was not absorbed by the fish, but adsorbed on their skin.

Food Exposure

In feeding studies no consistent relationship was found between half-lives and degree of chlorination when 2,7-diCDD, 2,3-diCDD, 1,2,3,4-tetraCDD, octaCDD, 3,6-diCDF, and octaCDF were fed to

rainbow trout in one single dose (Niimi and Oliver, 1986). Half lives in days for the CDDs and CDFs ranged from 2 to 43 and 7 to 12, respectively (Table 3.13). Absorption efficiencies were low, ranging from 2 to 16 percent. However, 2,7-diCDD, which has a structure similar to 2,3,7,8-tetraCDD, had greater than 30 percent assimilation, a finding that suggests absorption through the gut might be influenced by the steric configuration of these compounds.

In two other longer term feeding studies, Kleeman et al. (1986a and 1986b) exposed rainbow trout and yellow perch to a diet of 494 pg/g of radiolabelled 2,3,7,8-tetraCDD for 13 weeks and observed half-lives of 105 and 126 days, respectively.

ACUTE AND CHRONIC TOXICITY

According to EPA (1984a), available fish and invertebrate acute and chronic toxicity data for 2,3,7,8-tetraCDD are too limited to permit derivation of water quality criteria. However, the available studies can give useful indications of toxicity. The majority of acute and chronic toxicity investigations have studied only the toxicity of 2,3,7,8-tetraCDD to freshwater species. Most studies have been static or static renewal bioassays. Only one acute bioassay has been reported involving CDDs and CDFs other than 2,3,7,8-tetraCDD. However, this test included exposure to several congeners simultaneously, and it is unclear which congener caused the reported toxicity. Few chronic toxicity studies exist from exposure to CDDs and CDFs. Polychlorinated dibenzofurans mainly have been studied in terms of chronic oral toxicity (e.g., feeding studies).

Acute Toxicity

Fish: Delayed Effects

A majority of the reported 2,3,7,8-tetraCDD acute toxicity studies on aquatic organisms have shown a pattern of delayed effects, mimicking the expected response time of low level, long term exposures. This same unusual action pattern has also been observed in acute exposures of 2,3,7,8-tetraCDD to mammals (McConnell et al., 1978). Due to this delay, 96-hour acute tests with 2,3,7,8-tetraCDD are typically followed by long observation periods of up to 24 weeks.

Miller et al. (1973) observed in 96 hour static exposure tests with coho salmon and guppies that initial responses did not occur for 5 to 10 days after the exposure period and mortality often extended over the next 2 months. Helder (1981) exposed juvenile

TABLE 3.13

HALF-LIVES OF CDDs IN RAINBOW TROUT
AFTER A SINGLE ORAL EXPOSURE
(Adapted from Niimi and Oliver, 1986)

COMPOUND	ORAL EXPOSURE CONCENTRATION (ug/l oil)	HALF-LIFE (Whole Body) (Days)
2,7-diCDD	82	2
2,3-diCDD	37	7
1,2,4-triCDD	38	12
1,2,3,4-tetraCDD	30	43
octaCDD	30	15
3,6-diCDF	115	12
octaCDF	15	7

rainbow trout to 100 ppt 2,3,7,8-tetraCDD for 96 hours and observed a sudden increase in mortality after the 21st day; by the 27th day, all trout had died.

Helder (1980 and 1981) exposed rainbow trout and pike eggs to concentrations of 0.1 to 10.0 ng/l 2,3,7,8-tetraCDD for 96 hours. Observation for mortality continued through three life stages: egg, yolk sac fry, and feeding fry. For both species, significant mortality occurred in the yolk sac fry stage at 1.0 and 10.0 ng/l (Table 3.14). The author suggested that the highly lipophilic character of 2,3,7,8-tetraCDD may be the cause of the high mortality to yolk sac fry. This stage may be vulnerable because, as demonstrated in vitro by other lipophilic compounds, 2,3,7,8-tetraCDD may readily accumulate in the triglyceride fraction of the yolk and become mobile several days after hatching when the fry utilize the triglycerides in the yolk as food.

TABLE 3.14

PERCENT MORTALITY OF RAINBOW TROUT AND NORTHERN PIKE FRY WITH YOLK SAC EXPOSED TO 2,3,7,8-TETRACDD (Data from Helder, 1980 and 1981)

	EXPOSURE CONCENTRATIONS (ng/l)				
	Acetone Control	Control	0.1	1.0	10.0
Rainbow trout	0.9	0.8	0.9	2.3[a/]	15.8[a/]
Northern pike	14.0	9.2	14.2	48.8[a/]	94.1[a/]

[a/] $p>0.001$

Due to the delayed lethality normally found in 2,3,7,8-tetraCDD bioassays, the expression of LC_{50} values as the concentration of toxicant giving 50 percent mortality at the end of a 96 hour exposure is not a meaningful indicator of 2,3,7,8-tetraCDD toxicity. As a result, the literature reports modified LC_{50}s, measuring mortality at some given time after the exposure period. There is not yet agreement on a standardized post-exposure delay for the calculation of LC_{50}. Among 2,3,7,8-tetraCDD modified LC_{50} values reported for 96 hour exposure tests on fish are 100 ng/l at 21 days for juvenile rainbow trout (Helder, 1981) and 5.6 ug/l (LC_{55}) at 60 days for juvenile coho salmon (Miller et al., 1979). See Table 3.15 for a summary of 2,3,7,8-tetraCDD bioassays. These data are presented in Figure 3.2.

TABLE 3.15

EFFECTS OF 2,3,7,8-TETRACDD ON AQUATIC ORGANISMS
(Adapted from Kenaga and Norris, 1983)

SPECIES	TEST DURATION[a/]	EFFECT	CONC. IN WATER (ng/l)	REFERENCE
Snail				
Physa sp.				
(adult)	36d/48d	reduced reproduction	200	Miller et al., 1973
Oligochaete worm				
Paranais sp.				
(adult)	55d/55d	reduced reproduction	200	" " "
Mosquito				
Aedes aegypti				
(larvae)	17d/39d	No effect on pupation	200	" " "
Guppy				
Poecilia reticulat				
(9 - 40mm)	5d/37d	Feeding decline, skin discoloration, fin necrosis, mortality	10000	" " "
"	5d/5d	LC8	100	Norris & Miller, 1974
" " "	5d/21.7d	LC50	100	"
" " "	5d/11.6d	LC50	1000	"
" " "	5d/18.2d	LC50	10000	"
" " "	5d/37d	LC100	100	"
Coho salmon				
Oncorhynchus kisutch				
(juvenile)	24-96h/40d	LC100	56	Miller et al., 1973
"	96h/60d	LC55	5.6	" " "
"	96h/60d	No effect on feeding, growth, and survival	0.56	Miller et al., 1979
Mosquitofish				
Gambusia affinis				
(gravid)	8d/15d	LC100	2.4-4.2	Yockim et al., 1978

TABLE 3.15 (continued)

EFFECTS OF 2,3,7,8-TETRACDD ON AQUATIC ORGANISMS
(Adapted from Kenaga and Norris, 1983)

SPECIES	TEST DURATION[a/]	EFFECT	CONC. IN WATER (ng/l)	REFERENCE
Northern pike				
Esox lucius				
(eggs)	96h/23d	LC26	0.1	Helder, 1982
"	96h/23d	LC53	1	" "
"	96h/23	LC99	10	" "
Rainbow trout				
Salmo gairdneri				
(juvenile)	64h/72d	LC12	10	Helder, 1981
"	64h/21d	LC50	100	" "
"	64h/27d	LC100	100	" "
"	64h/73d	EC54 - growth	10	" "
American bullfrog				
Rana catesbeiana				
adults [b/]	-- /35d	No mortality	500[c/]	Beatty et al., 1976
tadpoles	"	" "	1000[c/]	" " " "
Alga				
Oedogonium				
cardiacum	32d/32d	No effect	1330	Isensee, 1978

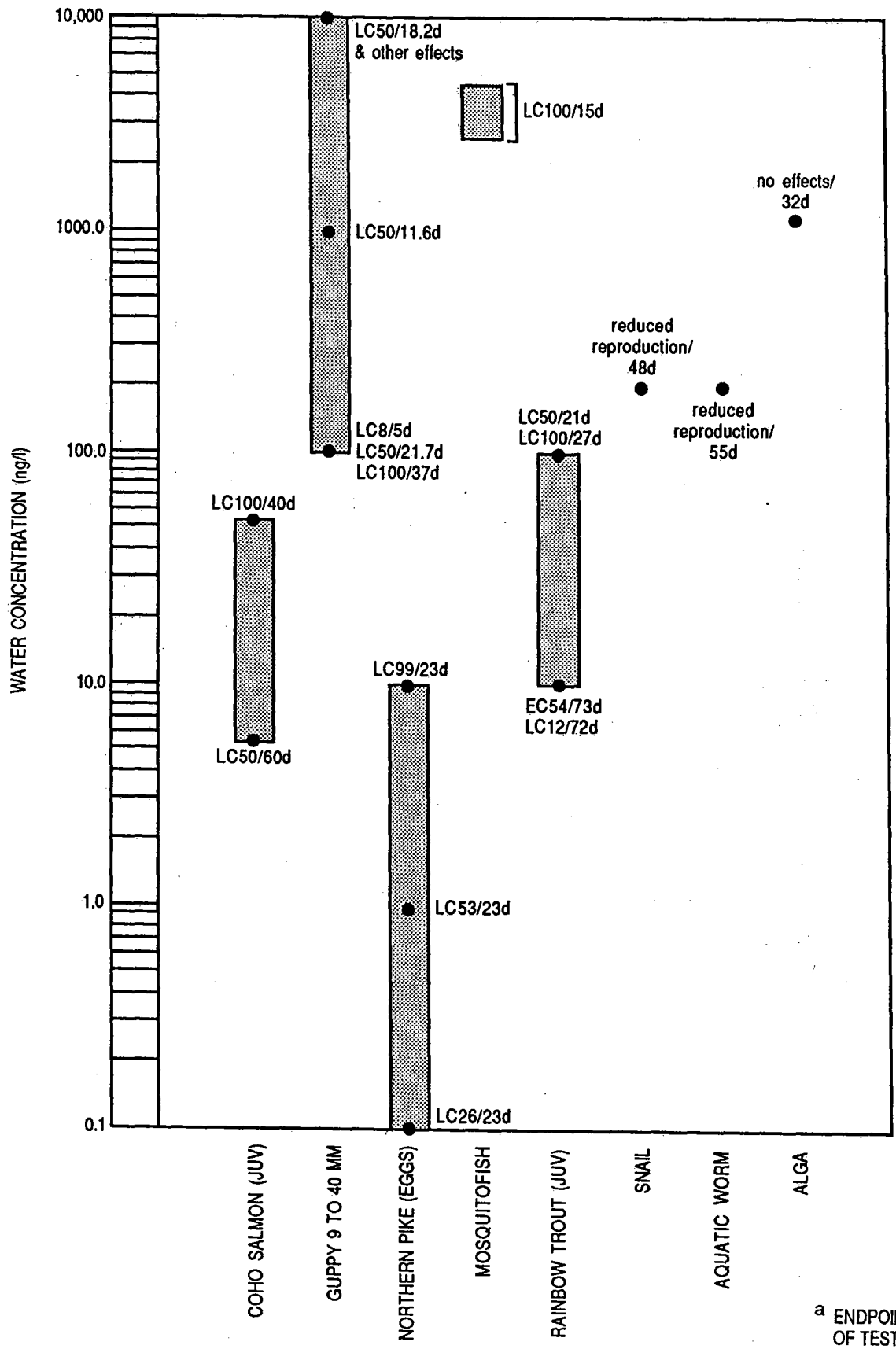
a/ Duration of exposure/post exposure observation period

b/ One intraperitoneal injection

c/ Dosage expressed in ug/kg of organism or food

Figure 3.2

RANGE OF 2,3,7,8-TETRACDD CONCENTRATIONS TOXIC TO AQUATIC SPECIES [a]



[a] ENDPOINT/DURATION OF TEST IN DAYS

Fish: Growth Effects

The most common effect reported in the 2,3,7,8-tetraCDD bioassay literature was growth retardation for several species of fish. For rainbow trout yolk sac fry exposed as eggs to 0.1, 1.0, and 10 ng/l of 2,3,7,8-tetraCDD for 96 hours, significant growth retardation occurred at all levels of exposure (Figure 3.3). At the 0.1 and 1.0 ng/l exposure concentrations, reduced growth was not significant until 72 and 118 days, respectively, after fertilization (exposure began just after fertilization). However, at the 10 ng/l concentration, growth retardation occurred throughout the entire experiment (Helder, 1981). In the same study, yolk sac fry were also exposed to 2,3,7,8-tetraCDD at 1.0 ng/l and juveniles at 10 and 100 ng/l. Significant growth retardation occurred at all concentrations during the entire experiment. Pike fry exposed to 2,3,7,8-tetraCDD in the egg stage for 96 hours to 0.1, 1.0, and 10 ng/l showed significantly shortened body lengths for a long period of time after hatching (Helder, 1980) (Figure 3.4). The growth of coho salmon was markedly inhibited by the 80th day after a 96-hour exposure to 56 ng/l 2,3,7,8-tetraCDD (Miller et al., 1973) (Figure 3.5).

Fish: Histopathology

Several acute toxicity studies on fish have found histopathological effects such as fin necrosis, loss or underdevelopment of caudal fins, edema, liver necrosis, and hemorrhaging from exposures to 2,3,7,8-tetraCDD ranging from 0.1 ng/l to 10 ug/l (Norris and Miller, 1974; Helder, 1980, 1981 and 1982; Miller et al., 1973 and 1979). Edema, often generalized, was the most consistent syndrome among several species.

Helder (1980 and 1981) conducted extensive studies on the histopathological effects of 2,3,7,8-tetraCDD on rainbow trout eggs, yolk sac fry and juveniles and on pike eggs. For the eggs of both species exposed to 1.0 and 10 ng/l of 2,3,7,8-tetraCDD, generalized hemorrhaging and edema increased with increasing dose. Degenerative changes and necrosis in the liver parenchymal cells were also observed. Newly hatched rainbow trout yolk sac fry exposed to 1 ng/l 2,3,7,8-tetraCDD were affected in the same manner. The exposed rainbow trout eggs that survived to the fry stage (12 weeks) developed shortened maxillas and opercular defects.

Figure 3.3
EFFECTS ON RAINBOW TROUT WEIGHT
AFTER 96 HR. EXPOSURE TO 2,3,7,8-TetraCDD
(Adapted from Helder, 1981)

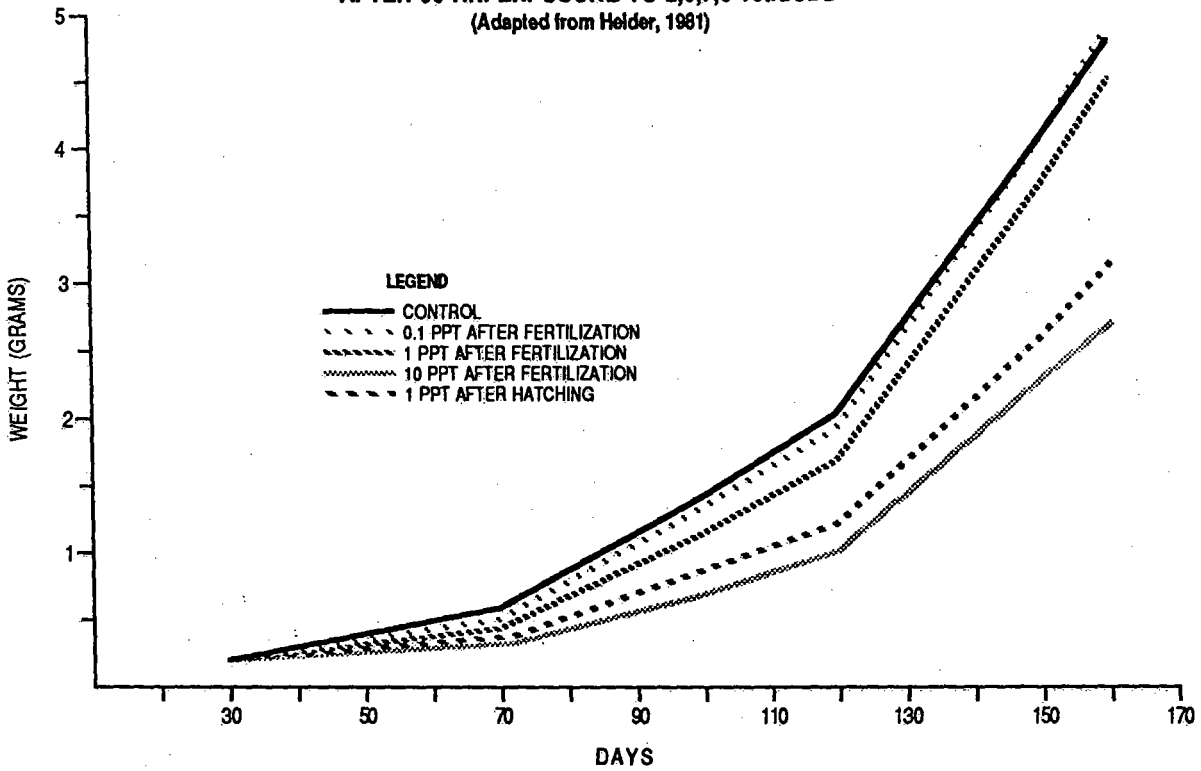


Figure 3.4
EFFECTS ON PIKE BODY LENGTH
AFTER 96 HR. EXPOSURE TO 2,3,7,8-TetraCDD
(Adapted from Helder, 1980)

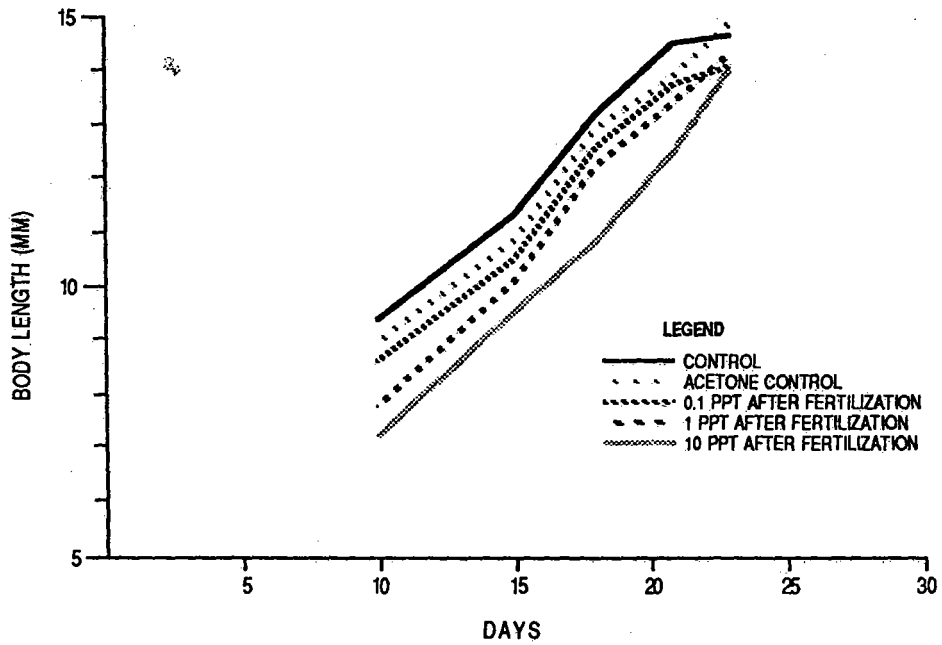
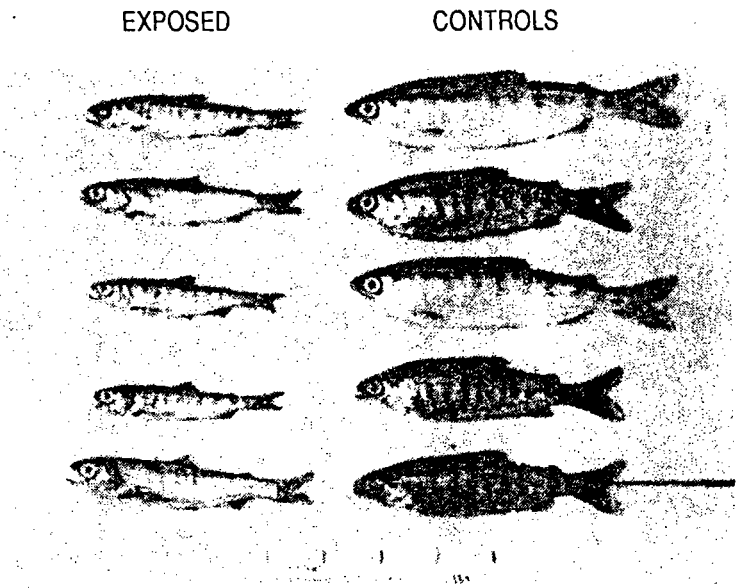


Figure 3.5

EFFECTS ON COHO SALMON GROWTH AT 80 DAYS
AFTER A 96 HR. EXPOSURE TO 56 ng/l OF 2,3,7,8-TetraCDD

(Miller et al.,1973)



Invertebrates

According to EPA (1984a), there are no data available to calculate 48 or 96 hour LC_{50} or EC_{50} values for invertebrate exposures to any CDD or CDF.

Amphibians

One study examined the effects of 2,3,7,8-tetraCDD on the larval and adult American bullfrog, Rana catesbeiana (Beatty et al., 1976). Intraperitoneal injections of up to 1 mg 2,3,7,8-tetraCDD/kg of body weight showed no effect on either lifestage during 35 observation days. Although this method of toxicant administration is not comparable to published studies on fish, the study suggests that Rana may be less sensitive to 2,3,7,8-tetraCDD.

Chronic Toxicity

Fish

Few chronic toxicity studies from exposure through the water medium are reported in the literature. Oral chronic toxicity studies have been conducted with 2,3,7,8-tetraCDD on rainbow trout, and with chlorinated dibenzofurans on Atlantic salmon and brook trout.

In a study by Yockim (1978) to observe the toxicity and environmental fate of 2,3,7,8-tetraCDD, mosquitofish (Gambusia affinis) were exposed to 2,3,7,8-tetraCDD concentrations in water ranging from 2.4 to 4.2 ng/l. All fish died after 15 days exposure. Gravid mosquitofish exposed to the same concentrations for 8 days lost all external signs of pregnancy and died after 15 days. The gravid control fish remained visibly pregnant and at autopsy showed fry in the late stages of development.

Hawkes and Norris (1977) fed young rainbow trout 2,3,7,8-tetraCDD in dried fish food at levels of 2.3 ppt, 2.3 ppb, 2.3 ppm, equivalent to an intake level of 0.0000064, 0.0072, and 4.2 ug 2,3,7,8-tetraCDD/kg body wet weight/day, respectively (calculated by Kenaga and Norris (1983) assuming wet weight is 5 times dry weight). Levels of 0.0000064 and 0.0072 ug/kg had no effect on food consumption, growth, or survival. However, when fish were exposed to 4.2 ug/kg, 50 percent mortality occurred after 61 days and 96 percent mortality by 71 days. At 4.2 ug/kg other effects were observed, including decreased feeding, growth reduction, fin erosion, and changes in liver tissue. These effects are quite similiar to those seen in fish exposed to 2,3,7,8-tetraCDD in water.

There is little information on the chronic toxicity of specific higher chlorinated dibenzofurans. Currently available data include very brief reports on chronic toxicity studies of fish fed CDF contaminated food.

Zitko and Choi (1973) reported that juvenile Atlantic salmon fed dry fish food spiked with mixed di-, tri-, tetra-, and octaCDFs in concentrations of 2.1, 4.4, 2.2, and 9.7 ppm respectively, for up to 140 days showed median mortality at 120±30 days. Only octaCDF was found in the tissues of the salmon.

However, Zitko et al., (1973) found no mortality when immature brook trout were fed several doses of 2,8-diCDF totaling 107 to 361 ug/g wet weight for 50 days. No mortality resulted, even after administration of a single dose as high as 122 ug/g.

Invertebrates

Miller et al. (1973) conducted long term static bioassays on snails (Physa sp.), mosquito larvae (Aedes aegypti), and aquatic worms (Paranais sp.). At exposures of 0.2 ug/l 2,3,7,8-tetraCDD, the mosquito larvae were not affected, but snails and worms both showed reduced reproductive success.

Aquatic Plants

Aquatic plants appear to be insensitive to low concentrations of 2,3,7,8-tetraCDD. No attempts have been made to determine the maximum no-effect levels (Kenaga and Norris, 1983). The limited existing data are from microcosm studies in which an alga, Oedogonium cardiacum, was not affected in a 31 day exposure to 2,3,7,8-tetraCDD concentrations ranging from 2.4 to 4.2 ng/l (Yockim et al. 1978). In a separate study, O. cardiacum was not affected in higher 2,3,7,8-tetraCDD concentrations of up to 1330 ng/l for a 31 day exposure (Isensee and Jones, 1975; Isensee, 1978).

Mechanisms of Action

Information on CDD and CDF mechanisms of action is lacking. A few, very general and brief, discussions focus only on the 2,3,7,8-tetraCDD congener. In a discussion on the sites and mechanisms of toxicity of 2,3,7,8-tetraCDD, Norris and Miller (1974) noted that delayed mortality in guppies is consistent with the hypothesis that 2,3,7,8-tetraCDD induces liver dysfunction; such dysfunction has been shown in tests with rodents. However, Helder (1982) suggests that his observation of hemorrhaging and

edema within the eggs of rainbow trout 21 days after exposure to 2,3,7,8-tetraCDD indicates a mechanism other than hepatic damage, because the liver at this point is just beginning to develop. He suggests the damage in this case is probably vascular.

SUMMARY AND DISCUSSION

Bioconcentration

Bioconcentration factors have not been reported in the literature for chlorinated dibenzofurans. Measured BCFs for 2,3,7,8-tetraCDD and other chlorinated dibenzo-p-dioxins are lower than would be expected from their high K_{ow} (octanol/water partition coefficient) values. However, most of the bioconcentration and bioaccumulation studies reviewed did not determine BCFs after a steady state had been reached. Thus, short-term laboratory exposures may give underestimates of potential BCFs for organisms exposed in the environment for long periods of time. Limited membrane transport of CDDs also may result in lower BCFs than expected. This limitation could be due in part to their large molecular size, high K_{ow} values and low water solubilities, resulting in binding to particulates and dissolved organic carbon (DOC). Congeners associated with particulates or dissolved organic carbon tend to show concentrations in the water higher than what may actually be available to non-filter feeding organisms.

Many of the studies reviewed used radiolabelled ^{14}C CDDs to determine bioconcentration factors. This method could result in overestimation of CDD concentrations in water and therefore lower BCFs. This is because the measured radioactivity may include degradation products if actual chemical species are not analytically identified.

Steric configurations such as the planar structure of 2,3,7,8-tetraCDD can affect the rate of membrane transport. The BCF for 2,3,7,8-tetraCDD is higher than that for any other congener. Fish and invertebrates have been shown to concentrate 2,3,7,8-tetraCDCD in their tissues up to approximately 9000 times the concentration in water. Aquatic organisms exposed to mixtures of many different CDDs and CDFs in water tend to accumulate 2,3,7,8-chlorinated congeners. However, non-2,3,7,8-chlorinated congeners, in the absence of 2,3,7,8-chlorinated congeners, can be accumulated in aquatic organisms.

Distribution of 2,3,7,8-tetraCDD in fish tissue follows what would be expected from its high octanol to water partition coefficient. The greatest proportion of the accumulated 2,3,7,8-tetraCDD is associated with the fatty tissue of aquatic organisms exposed to contaminated water.

Compared with research using rodents and mammals on the metabolism of CDDs, very little is known about CDD metabolism in aquatic species. Glucuronide conjugates found in the bile of yellow perch and rainbow trout indicate biotransformation, but the mechanism is not known.

Long-term water exposures approaching equilibrium show that the retention time of 2,3,7,8-chlorinated congeners in tissues of aquatic organisms increases with increasing chlorination. The effect that longer retention of more highly chlorinated congeners has on toxicity is uncertain due to the lack of aquatic toxicity data for congeners other that 2,3,7,8-tetraCDD.

Toxicity

Two unusual aspects of 2,3,7,8-tetraCDD's toxicity which make it unique are (1) its pattern of delayed effects after acute exposures and (2) the inordinately low concentrations (as low as 0.1 ng/l) which cause a toxic reaction in aquatic organisms.

Growth retardation was the most commonly reported effect of several species of fish yolk sac fry after 96-hour exposures to 2,3,7,8-tetraCDD. Histopathological effects included fin necrosis, edema, liver necrosis, and hemorrhaging. Edema, the most consistently reported syndrome, may be the result of induced liver dysfunction or vascular damage observed in fish exposed to 2,3,7,8-tetraCDD.

Aquatic plants tested at low exposures appear to be insensitive to 2,3,7,8-tetraCDD. The effects of other CDD and CDF congeners on aquatic organisms are unknown. Studies are needed to determine toxicity of these compounds because these compounds have been detected in aquatic environments and they have been found to bioaccumulate in aquatic organisms.

As this report went to press, a recent study described adverse effects of 2,3,7,8-tetraCDD and 2,3,7,8-tetraCDF at even lower concentrations than previously reported (Mehrle et al., 1988).

This chronic study of rainbow trout was a 56-day flow-through experiment consisting of 28 days of exposure followed by 28 days of depuration. At 38 parts per quadrillion 2,3,7,8-tetraCDD, the lowest concentration tested, significant effects were observed on growth and survival. Because effects were observed at the lowest level tested, a no observed effect concentration (NOEC) could not be derived.

The CDF also was very toxic. At 0.9 parts per trillion (ppt) 2,3,7,8-tetraCDF, growth was adversely affected; survival was reduced at 4 ppt. NOEC values were 0.4 ppt for growth and 1.8 ppt for survival. During the 28-day depuration period, mortality continued and there was "no apparent recovery in clean water" in both the CDD and the CDF experiments. In addition to survival and growth, the authors monitored five behavioral changes: reduced feeding, lethargic activity, unresponsiveness, resting on the bottom, and head-up swimming.

The same study determined bioconcentration factors of 39,000 for 2,3,7,8-tetraCDD and 6,049 for 2,3,7,8-tetraCDF, factors higher than previously described. Mehrle et al. (1988) concluded that 2,3,7,8-tetraCDD is more than 10,000 times as toxic to fish as the insecticides endrin or toxaphene and that 2,3,7,8-tetraCDF is roughly 1,000 times as toxic.

CHAPTER 4: MAMMALIAN TOXICOLOGY

Adverse health effects from exposure to CDD and CDF compounds have been the subject of intense study in recent years. The 2,3,7,8-tetraCDD congener has been the most extensively studied CDD or CDF compound and, being the most toxic of either class, is the standard for comparison of toxic effect. The principal effects include high acute toxicity, immunotoxicity, teratogenicity, adverse effects on reproduction, enzyme induction, chloracne, carcinogenicity, and possibly mutagenicity. Adverse health effects related to CDDs and CDFs have been recently reviewed by CARB and CDHS (1986), U.S. EPA (1985b), NRCC (1981, 1984), and Huff et al. (1980).

ABSORPTION AND TISSUE DISTRIBUTION

Gastrointestinal absorption and tissue distribution studies have been done on a limited number of CDD and CDF compounds, mostly on 2,3,7,8-tetraCDD and 2,3,7,8-tetraCDF. From 50 to 86 percent of the administered dose of 2,3,7,8-tetraCDD is absorbed from the rat gastrointestinal tract. The carrier used to administer the dose also affects the extent of absorption, with organic solvent mixtures, such as a combination of corn oil and acetone, producing the greatest effect. When 2,3,7,8-tetraCDD was administered to rats in combination with activated charcoal, very little of the dose was absorbed. A decrease in the absorbed dose was seen when 2,3,7,8-tetraCDD was given in combination with soil; the dose absorbed decreased as the length of time the 2,3,7,8-tetraCDD was in contact with the soil increased (U.S. EPA, 1985b).

Umbreit et al. (1986) used guinea pigs to study the bioavailability of 2,3,7,8-tetraCDD from contaminated soil obtained from a site where the herbicides 2,4,5-T and 2,4-D were manufactured (Newark, N.J.), and from a second site where the chemical stills from this plant were dismantled for salvage. Animals of both sexes were dosed by gavage with a soil suspension at levels of 6 ug/kg and 12 ug/kg of 2,3,7,8-tetraCDD. No toxicity was produced by soils from either site. The bioavailability of 2,3,7,8-tetraCDD from the manufacturing site soil was less than 0.5 percent, and from the salvage site soil was 21.3 percent.

McConnell et al. (1984) administered a soil suspension from two sites contaminated with 2,3,7,8-tetraCDD along with other CDDs and CDFs to both rats and guinea pigs by gavage at levels of 1, 3, or 10 ug/kg of 2,3,7,8-tetraCDD. This soil was obtained from the Times Beach, Missouri area and was contaminated when waste oil containing CDDs and CDFs was applied to roads to

control dust. Signs of acute toxicity, induction of aryl hydrocarbon hydroxylase activity and measurable levels of 2,3,7,8-tetraCDD in various tissues were seen. The bioavailability of 2,3,7,8-tetraCDD from these soils was estimated at about 85 percent by Umbreit et al. (1986), and at about 25 to 50 percent by Lucier et al. (1986).

Absorption of 2,3,7,8-tetraCDF occurs readily from the GI tract in guinea pigs, rats and mice. In mice 70 to 90 percent of the dose is absorbed depending on the strain used. In guinea pigs absorption has been about 90 percent of the dose given by gavage (NRCC, 1984). Skin absorption of 2,3,7,8-tetraCDD has been estimated at about 40 percent of an equivalent oral dose in rats. This estimate assumes that levels in the liver can be used to estimate the amount absorbed by both oral and dermal routes (U.S. EPA, 1985b).

Tissue distribution studies using 2,3,7,8-tetraCDD have shown that it has an affinity for tissues with high lipid content, which is not surprising due to its lipophilic nature. In the rat, the liver accounted for 38 to 52 percent of a single dose seven days after dosing by both oral and intraperitoneal routes (U.S. EPA, 1985b). The rat, mouse, and hamster have similar distribution patterns, with the liver, then adipose tissue, having the largest percentage of the dose. Levels in other tissues are generally much lower. For non-human primates and guinea pigs, adipose tissue levels are higher than levels in the liver, with high levels also present in the skin (CARB and CDHS, 1986).

With 2,3,7,8-tetraCDF the route of administration did have some effect on distribution initially, but after 3 days it was similar for both the intravenous and oral routes. Greater than 95 percent of the tissue levels in the rat and guinea pig were believed to be unmetabolized 2,3,7,8-tetraCDF (NRCC, 1981). The greater amount of adipose tissue present in the female mouse relative to the male is thought to have produced a difference in the tissue distribution of 2,3,7,8-tetraCDD fed in the diet. Male mice stored approximately 15 percent more of the total body residue in the liver compared to females (U.S. EPA, 1985b).

Tissue distribution of CDDs in humans has been estimated from accidental exposures caused by industrial releases or contamination of food. One report describes a 55 year-old woman exposed to 2,3,7,8-tetraCDD as a result of an industrial accident in Seveso, Italy. This woman, who died seven months after exposure, also had a carcinoma not believed related to the accidental exposure which involved the pancreas, liver, and lungs. Levels were highest in the adipose tissue and the

pancreas, with lower levels in the liver. The effect of the neoplasms on the (normal) distribution of 2,3,7,8-tetraCDD is not known, but the pattern was similar to that seen in the guinea pig and non-human primates (NRCC, 1981; U.S. EPA, 1985b).

Movement across the placenta occurs with 2,3,7,8-tetraCDD. The placenta affects distribution to the fetus in mice, with higher levels found in the embryo before the placenta is in place compared to after. Levels in the placenta itself were found to be greater than those in the fetus by an order of magnitude. The fetal liver concentrated 2,3,7,8-tetraCDD to a lesser extent than did the maternal liver, with studies indicating distribution to other organs being similar to that seen in maternal tissues (U.S. EPA, 1985b).

In Japan, analysis of tissues from humans ingesting rice oil contaminated with various CDF isomer groups (Yusho poisoning) has provided some data on human tissue distribution in man. Of the total CDFs reported to be in the contaminated oil, approximately 2 percent were triCDFs, 22 percent tetraCDFs, 46 percent pentaCDFs, and 30 percent hexaCDFs, with at least 40 congeners of these isomer groups determined to be present. In human tissues analyzed, higher concentrations were found in adipose tissue compared to liver. The pentaCDFs and hexaCDFs were most persistent, in some cases being detected up to nine years after exposure. An inverse relationship appeared to exist between tissue concentrations and length of survival after exposure (NRCC, 1984).

In animals, retention of isomer groups may vary between species, and there are also differences between species in the organs where isomer groups are retained. The rat retained a greater proportion of the dose of the hexaCDF group in the liver compared to the monkey in a study by Kuroki et al. (1980). In this same study, the monkey retained the 2,3,4,7,8-pentaCDF isomer to a greater extent than the 1,2,4,7,8-pentaCDF or the 1,2,3,7,8-pentaCDF isomers in the liver, indicating the differential retention of isomers in the same organ for a given species.

The NRCC (1984) reported that CDFs crossed the placenta in small amounts relative to the maternal dose, with the mouse fetus accumulating the various isomer groups to different degrees when a mixture of CDFs was fed to female mice during gestation. This same study also found CDFs transferred to offspring in the milk to a greater extent than across the placenta when the same CDF mixture was fed to female mice during lactation.

METABOLISM AND ELIMINATION

Studies with labeled 2,3,7,8-tetraCDD in various animal species have demonstrated biotransformation of CDDs. In rats, hamsters, and guinea pigs, ^{14}C labeled 2,3,7,8-tetraCDD produced labeled glucuronide conjugates in the bile and sulfate conjugates in the urine (U.S. EPA, 1985b). In rats, 90 to 100 percent of the recovered radioactivity in the bile appeared to be metabolites of 2,3,7,8-tetraCDD. In hamsters and guinea pigs there was no unmetabolized ^{14}C labeled 2,3,7,8-tetraCDD found in either urine or bile (NRCC, 1981).

CDDs and other halogenated hydrocarbons appear to be metabolized in the liver by mixed function oxidases (MFO). The MFO system is a multi-enzyme group located in the endoplasmic reticulum of the cell. It metabolizes a wide range of substances from many different chemical classes. The MFOs are present in liver in high amounts. Animal studies have indicated metabolism of CDDs and CDFs in the liver by either cytochrome P-450, or more likely by cytochrome P_1-450, which are components of this enzyme system.

In rats, studies incorporating age and sex related differences in MFO activity, along with differences produced through the use of both inhibitors and inducers of MFO activity, have helped to define an inverse relationship between MFO activity and toxicity (Beatty et al., 1978). Studies in dogs have detected metabolites consistent with biotransformation to an epoxide intermediate resulting from MFO activity (U.S. EPA, 1985b).

The 2,3,7,8-tetraCDD isomer is a potent inducer of MFO activity, causing an increase in smooth endoplasmic reticulum. Like 3-methylcholanthrene (3-MC), administration of 2,3,7,8-tetraCDD apparently results in the induction of cytochrome P-448 (P_1-450) (Doull et al., 1980). Associated aryl hydrocarbon hydroxylase oxidative activity is also induced by polycyclic hydrocarbons such as 2,3,7,8-tetraCDD (Hodgson and Guthrie, 1980). The role metabolism plays in the toxicity of CDDs is not known. Although an epoxide intermediate has been suggested (CARB and CDHS, 1986), metabolism in the case of 2,3,7,8-tetraCDD seems to be mostly a detoxification process producing metabolites less toxic than the parent compound (U.S. EPA, 1985b).

When a mixture of CDF isomer groups was administered to mice, the groups were metabolized at different rates. Metabolism in the liver was rapid relative to adipose tissue in a study done by Morita and Oishi (1977) and summarized by NRCC (1984). The monkey metabolized 2,3,7,8-tetraCDF slower than the rat, with about four times as much label (consisting mostly of metabolites) in the feces as in the urine (NRCC, 1984). Guinea pigs, the most

sensitive species, excrete equal amounts of label in urine and feces, but at a much slower rate than the monkey or rat. About 90 percent of the urine excretion is in metabolite form in the guinea pig, with the radioactivity in feces showing little evidence of being metabolized in one study (NRCC, 1984).

Elimination in most species is predominantly via the feces (80 to 100 percent) with small amounts in the urine. An exception is the hamster, with excretion in the urine and feces being 41 percent and 59 percent respectively (NRCC, 1981; U.S. EPA, 1985b). The biological half-life for 2,3,7,8-tetraCDD in all species tested is about 10 to 40 days, and is nearly three times greater in the guinea pig than in the hamster. Within a species the half-life for three strains of mice was seen to vary by a factor of two, with the two strains having the shorter half-life also having about half the amount of adipose tissue as the strain with the longer half-life. A study in the monkey indicated a half-life for 2,3,7,8-tetraCDD in adipose tissue of about one year (U.S. EPA, 1985b).

For most species tested elimination seems to follow first-order kinetics, with the guinea pig possibly having a zero-order rate. In several studies with the mouse, rat, guinea pig, and hamster all radioactivity associated with administration of labeled 2,3,7,8-tetraCDD appeared in the urine and bile as metabolites. Such metabolites have not been found in the liver and fat tissues themselves, possibly because they are readily excreted as they are formed. Unmetabolized 2,3,7,8-tetraCDD has been found in feces from the hamster and rat, implying another route in addition to the bile for fecal elimination (U.S. EPA, 1985b).

2,3,7,8-TetraCDD has been found in rats during lactation. The milk is also a route of excretion in humans, with 2,3,7,8-tetraCDD, 1,2,3,7,8-pentaCDD, 1,2,3,4,7,8-hexaCDD, 1,2,3,6,7,8-hexaCDD, 1,2,3,7,8,9-hexaCDD, 1,2,3,4,6,7,8-heptaCDD, and octaCDD having been detected (U.S. EPA, 1985b).

In various species CDFs also have different rates of elimination, with the half-life ranging from less than two days in the rat to 20 days in the guinea pig. Morita and Oishi (1977) administered a mixture of CDF isomer groups to mice which produced an estimated half-life of about two weeks, with the different groups eliminated at different rates. Variations in the amount of adipose tissue between two strains of mice was thought to have produced a two-fold difference in the biological half-life of 2,3,7,8-tetraCDF, with the strain with more adipose tissue having the longer half-life (NRCC, 1984).

Blood samples taken at one and two year intervals from persons in Taiwan who had ingested rice oil contaminated with CDFs (Yusho poisoning) demonstrated an estimated half-life of greater than a year for the 2,3,4,7,8-pentaCDF and 1,2,3,4,7,8-hexaCDF isomers. Analysis of blood samples eleven years after a similar human exposure in Japan could still detect the 2,3,4,7,8-pentaCDF isomer (NRCC, 1984).

Poiger and Schlatter (1986) studied the pharmacokinetics of tritium labeled 2,3,7,8-tetraCDD in a 42 year old, 92 kg adult human volunteer. After fasting overnight, 105 ng of [1,6-^{3}H] 2,3,7,8-tetraCDD dissolved in corn oil was given orally, corresponding to a dose of 1.14 ng/kg. During the first three days after dosing 11.5 percent of the radioactivity was eliminated in the feces, with another 1.5 percent eliminated by the same route over the next four days. From days 7 to 125 an additional 3.5 percent of the dose was found in the feces. No label was detected in urine during the 125 days following dosing, with the exception of the first few urine samples which contained small amounts of radioactivity.

Samples of subcutaneous adipose tissue were taken two weeks before, and again at 13 and 69 days after exposure. These latter two samples contained 2,3,7,8-tetraCDD at levels of 3.09 $\pm$ 0.05 and 2.85 $\pm$ 0.28 ppt, respectively. The authors estimated that approximately 90 percent of the dose was sequestered in the adipose tissue. Low levels were detectable in the blood, with 0.13 pg/ml measured 2 hours after dosing, 0.03 pg/ml after 5 days, which fell to <0.02 pg/ml after 12 days. Based on elimination in the feces, a half-life of 2120 days (5.8 years) was calculated, with the data supporting elimination by first-order kinetics (Poiger and Schlatter, 1986).

ACUTE, SUBCHRONIC, AND CHRONIC EFFECTS

Animal Data

Most data on the toxicity of CDDs has been obtained using 2,3,7,8-tetraCDD, which is considered the most toxic CDD or CDF. There is wide variability in sensitivity between species to the toxic effects of CDDs and CDFs, as Tables 4.1 and 4.2 demonstrate. For 2,3,7,8-tetraCDD the guinea pig is the most sensitive species tested, with an LD_{50} for the male of 0.6 ug/kg, and the hamster the least sensitive, with an LD_{50} for the male of up to 5,051 ug/kg.

Toxic effect after exposure to 2,3,7,8-tetraCDD is apparently more related to the total dose received than to whether the total dose is given all at once, or is distributed over time. Even

TABLE 4.1

COMPARATIVE SINGLE ORAL DOSE LD_{50} VALUES FOR CDD CONGENERS

(CARB AND CDHS, 1986)

Oral LD50 Values (ug/kg)

Chlorodibenzodioxin	Guinea Pig	Mouse	Rat	Monkey	Hamster	Rabbit	Dog
2,3,7,8-Tetra	0.6-2	14-284	22-45	70	1157-5051	115	>300,<3,000
Unsub		>50,000	>1,000,000				
2,3-Di			>1,000,000				
2,7-Di		>2,000,000	>1,000,000				
2,6-Di	>300,000	847,000,000	>5,000,000				
1,3,7-Tri		>15,000,000	>5,000,000				
2,3,7-Tri	29,444	>3,000	>1,000,000				
1,2,3,4-Tetra			>1,000,000				
1,3,6,8-Tetra	>15,000,000	>2,987,000	>10,000,000				
1,2,3,7,8-Penta	3.1	337.5					
1,2,4,7,8-Penta	1,125	>5,000					
1,2,3,4,7,8-Hexa	72.5	825					
1,2,3,6,7,8-Hexa	70-100	1,250					
1,2,3,7,8,9-Hexa	60-100	>1,440					
1,2,3,4,6,7,8-Hepta	>600						
Octa		>4,000,000	>1,000,000				

TABLE 4.2

COMPARATIVE SINGLE ORAL DOSE LD_{50} VALUES FOR CDFs COMPARED TO 2,3,7,8-TetraCDD (CARB AND CDHS, 1986)

Oral LD_{50} Values (ug/kg)

Chlorodibenzodioxin/ furan	Guinea Pig	Mouse	Rat	Monkey	Hamster	Rabbit	Dog
2,3,7,8-TetraCDD	0.6-2	114-284	22-45	70	1157-5051	115	>300,<3,000
Chlorodibenzofuran							
2,8-DiCDF		>15,000,000	>15,000,000				
2,4,8-TriCDF		>15,000,000	>5,000,000				
2,3,7,8-TetraCDF	5-10	>6,000	>1,000	1,000			
2,3,4,7,8-PentaCDF	<10						
2,3,4,6,7,8-HexaCDF	120						

with acutely toxic exposures, death is delayed, and may take from 5 to 45 days (U.S. EPA, 1985b). Weight loss occurs during this time, and is often described as "wasting away", with the animal apparently not able to utilize nutrients from the diet (CARB and CDHS, 1986).

2,3,7,8-TetraCDD-induced liver damage is seen in most species as necrosis, lipid accumulation, bile duct hyperplasia and an increase in liver to body weight ratio. Also in the liver, mixed function oxidase (MFO) activity is increased. In the rat a single dose of 200 ug/kg will produce liver necrosis, with 5 to 25 ug/kg causing fatty changes and an increase in both hepatic endoplasmic reticulum and MFO activity. In the mouse porphyria may also be seen (U.S. EPA, 1985b). This liver damage is generally not seen in the guinea pig, the most sensitive species tested (CARB and CDHS, 1986).

The immune system is affected in all species tested, with thymic atrophy caused by loss of cortical lymphocytes being the principal change (McConnell, 1980). The spleen, lymph nodes, and bone marrow may also be affected. Cell-mediated immunity is suppressed, with decreased resistance to bacterial infection demonstrated in animals exposed to 2,3,7,8-tetraCDD. It is thought that 2,3,7,8-tetraCDD may bind to the T-lymphocyte cell membrane, interfering with antigen and cell-cell recognition (U.S. EPA, 1985b).

Humoral immune response has also been reduced by 2,3,7,8-tetraCDD, with a decrease in antibody production and altered serum immunoglobulin levels detected in mice. These effects have been seen with a dose of 2,3,7,8-tetraCDD as low as 0.04 ug/kg/wk (Table 4.3). There is some indication that this immuno-suppression may be reversible with time (Poiger and Schlatter, 1983).

Kerkvliet et al. (1985) used C57B1/6 mice dosed by gavage in a study of humoral immune response using technical grade PCP and its commonly occurring contaminants, which include polychlor-inated diphenyl ethers, phenoxyphenols, dibenzodioxins and dibenzofurans. Several purified CDD and CDF congeners were also tested. A single oral dose was given two days before a challenge by sheep red blood cells, and five days later the peak splenic IgM antibody reponse was measured. Where technical grade PCP (86 percent PCP) produced a dose-related decrease in antibody response, analytical grade PCP (>99 percent PCP) had no effect. A chlorinated phenoxyphenol fraction and a chlorinated diphenyl ether fraction produced no immunosuppression when given at levels likely to be found in technical PCP, while the CDD and CDF fraction produced significant immunosuppression. OctaCDD and

TABLE 4.3

SUMMARY OF THE COMPARATIVE TOXICITY OF 2,3,7,8-TetraCDD IN GUINEA PIGS, RATS, AND MICE (U.S. EPA 1985b)

SPECIES	Immunotoxicity (ug/kg/week)		Teratogenicity (ug/kg/day)		Reproduction (ug/kg/day)	
	LOEL	NOEL	LOEL	NOEL	LOEL	NOEL
RAT	5.0	1.0	0.125	0.03	0.01	0.001
MOUSE	1.0	0.5	1.0	0.1	INSUFFICIENT DATA	
GUINEA PIG	0.04	0.008	ND	ND	ND	ND

ROUTE OF ADMINISTRATION FOR ALL DOSES IS EITHER ORAL OR GAVAGE.

LOEL = LOWEST OBSERVABLE EFFECT LEVEL

NOEL = NO OBSERVABLE EFFECT LEVEL

ND = NO DATA

several purified phenoxyphenol isomers also had no effect on immune response. The ID_{50} dose (that dose producing a 50 percent suppression in humoral immune response compared to controls) was 83 ug/kg for technical PCP, 7.1 ug/kg for 1,2,3,6,7,8-hexaCDD, 85 ug/kg for 1,2,3,4,6,7,8-heptaCDD, and 208 ug/kg for 1,2,3,4,6,7,8-heptaCDF. For comparison, the authors calculated an ID_{50} for 2,3,7,8-tetraCDD of 0.65 ug/kg from data produced by Vecchi et al. (1980) in a similiar study.

When 10 ppm technical PCP was fed to both Ah-responsive C57B1/6 and non-Ah-responsive DBA/2 mice for six weeks, immunosuppression and Ah-induced P_1-450 related enzyme activity were seen only in C57B1/6 mice. At a level of 250 ppm both strains showed signs of immunosuppression and enzyme induction, with the response significantly greater in C57B1/6 strain compared to the DBA/2. The authors suggest that these results support the conclusion that the immunosuppression produced by technical PCP is mediated by the CDD and CDF contaminants interacting with the Ah-receptor. Immunosuppression appeared to be additive in nature when 1,2,3,6,7,8-hexaCDD and 1,2,3,4,6,7,8-heptaCDD were given concurrently. When the linear dose-response curves for the congeners tested are compared they are parallel, which suggests a common mechanism of action for immunosuppression (Kerkvliet et al., 1985).

In subchronic studies with 2,3,7,8-tetraCDD using the mouse and rat, the liver was again the organ most affected. Rats dosed at 1 ug/kg/wk showed fatty changes of the liver, first seen 28 weeks into the study and persisting 12 weeks after dosing had ended. Elevated porphyrin levels in the liver have also been produced in rats dosed for 16 weeks, and remained high for up to six months after the exposure had ceased (U.S. EPA, 1985b).

In mice, subchronic exposure to 2,3,7,8-tetraCDD produced toxic hepatitis as the only effect. In female mice a NOEL of 2 ug/kg/wk was established for 2,3,7,8-tetraCDD. In male mice dosed at 1 ug/kg/wk, the lowest dose tested, toxic hepatitis was still apparent (U.S. EPA, 1985b). Chronic studies in mice and rats have also shown the liver to be the major target organ. In the rat, fatty infiltration of the liver is seen first, progressing to liver necrosis as the dose increases. A NOEL for the rat was established at 0.001 ug/kg/wk (U.S. EPA, 1985b). Chronic studies in the mouse and monkey have not established a NOEL for either species. Liver damage in mice has been seen at doses as low as 0.001 ug/kg/day. In the monkey, alopecia, edema, and pancytopenia has been produced at levels of 50 to 500 ppt in the diet (NRCC, 1981).

Human Data

Most observations on the toxicity of 2,3,7,8-tetraCDD to man indicate that the most common effects from exposure are chloracne, liver abnormalities, hematologic disorders, porphyria, and hyperpigmentation disorders (U.S. EPA, 1985b). Peripheral and central neurological disorders, seen as peripheral neuropathy, lethargy, and sensory impairment have also been reported (CARB and CDHS, 1986; NRCC, 1981). Most human exposures to CDDs and CDFs have occurred either occupationally or accidentally, and concurrently with exposure to other chemicals. In these situations the actual dose received could not be determined. It should be noted that in earlier literature, there were reports of severe liver disease and human fatalities associated with synthesis of 2,3,7,8-tetraCDD (May, 1973; Esposito et al., 1980). However, these brief observations have not been included in recent reviews.

Chloracne is the most common adverse effect seen in man after exposure to CDDs, and may occur anytime from a few days to weeks after exposure (U.S. EPA, 1985b). This dermal lesion is characterized by comedones and cysts, which may progress to pustules as the dose increases. It may subside within a few months or persist for years, with some cases lasting up to 15 years after exposure. 2,3,7,8-TetraCDD has produced chloracne in the monkey and rabbit, although it is usually not seen in other species (U.S. EPA, 1985b).

Human consumption of rice oil contaminated with polychlorinated biphenyls (PCBs) and CDFs in Japan (1968) and Taiwan (1979) produced a number of toxic effects known collectively as Yusho. In the Japan incident the contaminated oil was found to contain PCBs at levels of 1000 ppm and CDFs at levels of 5 ppm, with 2,3,7,8-tetraCDF detected at 0.45 ppm (Huff et al., 1980).

Adverse effects related to Yusho poisoning include:

- pigmentation disorders
- chloracne
- eye discharge
- swelling of upper eyelids
- distinctive hair follicles
- neurological disturbances

These effects are similar to those seen in experimental animals exposed to CDFs, and are generally attributed to the CDF contaminants in the oil. However, the presence of PCBs and polychlorinated quaterphenyls as contaminants in addition to the CDFs must also be considered (NRCC, 1984).

A recent study by Hoffman et al. (1986) examined persons living in an area of Missouri where waste oil contaminated with 2,3,7,8-tetraCDD was applied to roads to control dust. The exposed group had a mean residence time of 2.8 ± 1.9 years in an area where 2,3,7,8-tetraCDD levels in the contaminated soil ranged from 39 to 2200 ppb. Alterations of liver function, considered subclinical, consisted of a lower mean serum bilirubin level and an elevated mean urinary uroporphyrin level in the exposed group. A statistically significant increase in the serum levels of enzymes possibly associated with liver function was also seen as suggestive of a compound-related effect. An indication of depressed cell-mediated immunity, considered subclinical, was seen in the exposed group, but was not supported by a history of any increase in lengthy or recurring infections, or accompanied by clinical signs of immune suppression.

Defoliation efforts during the Vietnam War involved the use of about 19 million gallons of herbicides, of which 11 million gallons consisted of Agent Orange, a mixture of 2,4,5-T and 2,4-D (Wolfe et al., 1985). Application of the Agent Orange alone resulted in approximately 368 pounds of the 2,3,7,8-tetraCDD contaminant also being released. An epidemiological study by the United States Air Force utilizing a matched cohort design has examined the occurrence of adverse effects in Air Force personnel involved in the spraying operations.

Results indicate no relationship between herbicide exposure and any long-term health effects. However, the study did report many minor or indeterminate effects for which a cause-and-effect relationship could not be defined. The study design allows for annual mortality and other updates for an additional 20 year period for detection of any developing trends in mortality or disease (Wolfe et al., 1985).

In humans, a cumulative toxic dose is estimated at 0.1 ug/kg. Epidemiological studies, and data from persons exposed to chemical products contaminated with CDDs, indicate that adverse effects are variable in duration, and either may persist for years or subside (U.S. EPA, 1985b).

STRUCTURE-ACTIVITY RELATIONSHIPS

While most available data are related to 2,3,7,8-tetraCDD, enough work has been done on related compounds to indicate that certain biological activity associated with CDDs and CDFs appears to be related to molecular structure; the number and location of the chlorine atoms is particularly important. CDDs, CDFs and related halogenated aromatics seem to have a common mechanism of action for some effects, and are believed to be mediated by a common receptor. There are species differences in susceptibility to effects produced by CDDs and CDFs, and even though each species

tested does not exhibit exactly the same clinical signs, there are a number of effects that are commonly seen, including (U.S. EPA, 1985b; Goldstein, 1980):

1) progressive weight loss (wasting)
2) skin disorders
3) thymic atrophy
4) porphyria
5) enzyme induction
6) liver disorders
7) teratogenicity

2,3,7,8-TetraCDD is the most potent CDD or CDF producing these effects, with 2,3,7,8-tetraCDF the most potent among the CDFs.

CDD and CDF structures with chlorine atoms at the 2,3,7, and 8 positions are associated with the highest biological activity. Increasing or decreasing the number of chlorine atoms results in a decrease in activity. For example, octaCDD, chlorinated in all available positions, is considered to be essentially biologically inactive (U.S. EPA, 1985b). CARB and CDHS (1986) consider those CDDs and CDFs having four, five, six, or seven chlorine atoms, four of which are in the 2,3,7, and 8 positions to have potentially significant toxicity associated with them (Table 4.4).

TABLE 4.4
(CARB and CDHS, 1986)
CDDs AND CDFs OF TOXICOLOGICAL CONCERN

	Dibenzodioxins	Dibenzofurans
Tetrachloro	2,3,7,8	2,3,7,8
Pentachloro	1,2,3,7,8,	1,2,3,7,8 2,3,4,7,8
Hexachloro	1,2,3,4,7,8 1,2,3,6,7,8 1,2,3,7,8,9	1,2,3,4,7,8 1,2,3,6,7,8 1,2,3,7,8,9 2,3,4,6,7,8
Heptachloro	1,2,3,4,6,7,8	1,2,3,4,6,7,8 1,2,3,4,7,8,9

NOTE: The numbers indicate the position of chlorine atoms on the dibenzodioxin or dibenzofuran molecule.

Certain toxic effects of 2,3,7,8-tetraCDD and other CDDs and CDFs are believed to be at least partially mediated by binding to the Ah receptor, a soluble protein in the cytoplasm of the cell (Figure 4.1). According to this theory, after binding with the Ah receptor in the cell cytoplasm, the 2,3,7,8-tetraCDD-receptor complex moves into the cell nucleus in a manner believed similar to that suggested for steroid hormones (U.S. EPA, 1985a). In the nucleus this complex interacts with the Ah locus producing mRNAs during transcription. These mRNAs are used as templates by ribosomes during translation in the cytoplasm to produce the gene products of the Ah locus, which leads to increased levels of these products in the cell and any activity associated with them. Cytochrome P_1-450 levels along with aryl hydrocarbon hydroxylase (AHH) activity are increased, as is the level of Ah receptor in the cell. Examples of other increased enzyme activity apparently linked to the Ah locus (pleiotropy) include glutathione-s-transferase, choline kinase and ornithine decarboxylase (Roberts et al., 1985; Vickers et al., 1985; McKinney and McConnell, 1982).

The significance of this enzyme activity is to increase the biotransformation of not only CDDs and CDFs, but also that of other drugs and chemicals that are substrates for these enzymes. For many such chemicals, biotransformation may either increase toxicity through formation of a reactive intermediate, or decrease toxicity by formation of a less reactive product. Although as Figure 4.1 indicates, there is some uncertainty as to how CDDs and CDFs produce various biochemical effects (steps 9 to 11), the toxicity of 2,3,7,8-tetraCDD and other CDDs and CDFs appears to be unrelated to cytochrome P_1-450 or other metabolic activity in general (U.S. EPA, 1985b).

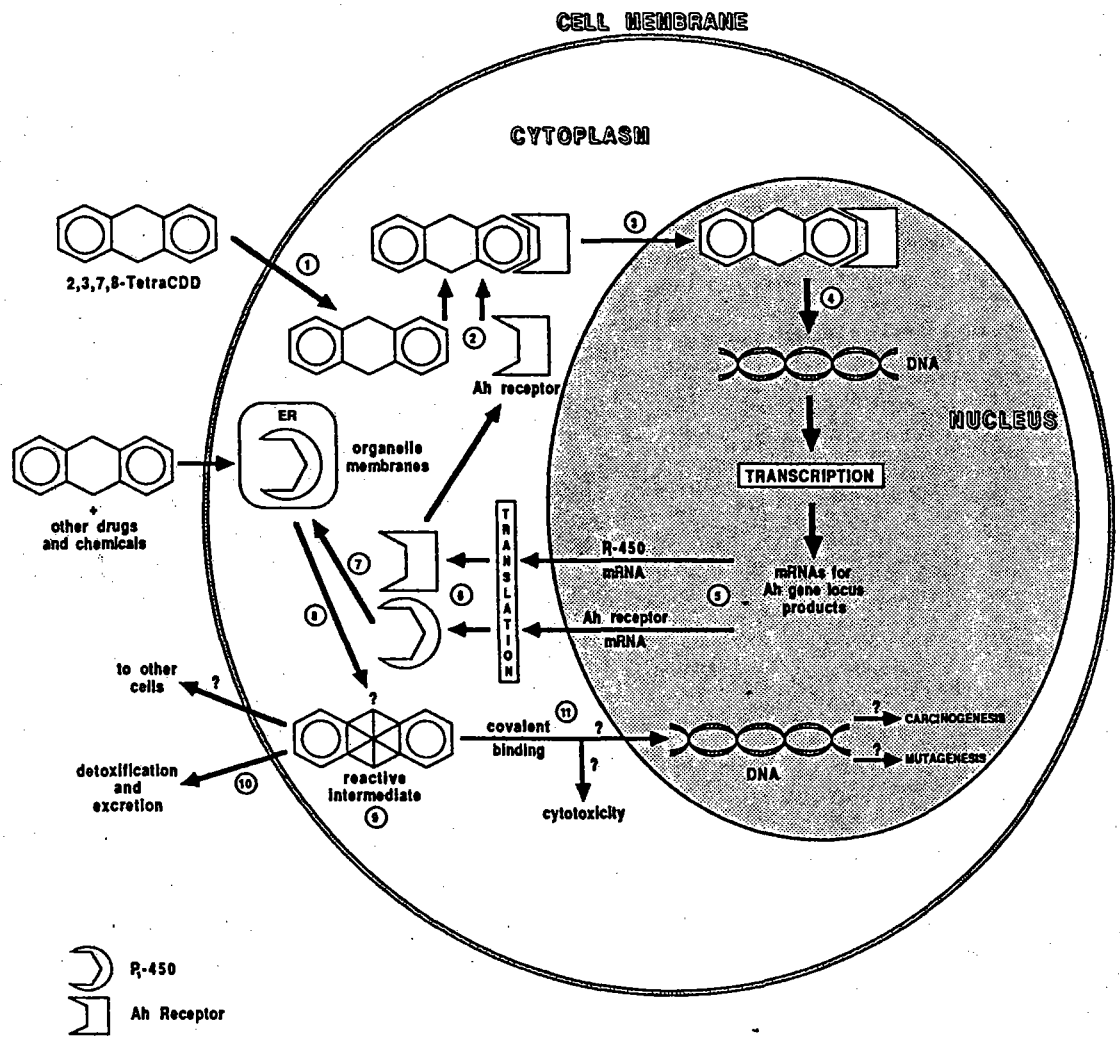
The tissue distribution of the Ah receptor has been determined in rodents, non-human primates and to a limited extent in humans. In these animal species, high concentrations of the receptor have been found in the liver, thymus, spleen, gastrointestinal tract, skin and pancreas. Although the Ah receptor has been determined to be present in human tissues such as the lung of a human fetus, in lung cells from adults, and in other cultured human cells, it is not known how widely distributed it is in the human population (Roberts et al., 1985).

2,3,7,8-TetraCDD is a very potent inducer of AHH activity, and is similar to, although more potent, than 3-methylcholanthrene and other halogenated aromatic compounds in this respect. Increased AHH activity has been seen not only in the hepatic endoplasmic reticulum, but also in other organelles such as the outer mitochondrial membrane and the nuclear membrane, in addition to other organs (Parkinson and Safe, 1981). Chemicals inducing AHH activity are usually competitive inhibitors of the binding of 2,3,7,8-tetraCDD to the receptor. The toxicity of such

FIGURE 4.1

MODEL FOR AH RECEPTOR MEDIATED MECHANISM OF ACTION

(Adapted from Roberts et al., 1985)



1. 2,3,7,8-TetraCDD passes through cell membrane
2. binds with Ah receptor in cytoplasm and is "activated"
3. activated 2,3,7,8-TetraCDD-receptor complex is transported to nucleus
4. complex interacts with DNA causing messenger RNAs (mRNAs) to be synthesized
5. mRNAs which code for gene products of the Ah locus eg, cytochrome P_1-450 and the Ah receptor, move into cytoplasm
6. the gene products coded for by the mRNAs are assembled by ribosomes
7. cytochrome P_1-450 is incorporated into organelle membranes, such as the endoplasmic reticulum, increasing the metabolizing capacity of the cell
8. the biotransformation of 2,3,7,8-TetraCDD and other substrate drugs and chemicals is increased
9. biotransformation products (metabolites) are formed which may be either more or less toxic than the original molecule
10. metabolites of many drugs and chemicals are detoxified and excreted, which appears to be the case with 2,3,7,8-TetraCDD
11. hypothesized actions of reactive intermediates in cytoplasm and nucleus

compounds, and also that of 2,3,7,8-tetraCDD does not always correlate with affinity for the receptor, or with the concentration of the receptor in the tissue (U.S. EPA, 1985b).

TERATOGENICITY AND REPRODUCTION

Studies with 2,3,7,8-tetraCDD have demonstrated teratogenic and fetotoxic effects in rats, mice, and rabbits, and fetoxicity in the monkey (CARB and CDHS, 1986). In mice the most common malformation seen is cleft palate, which has been produced with a dose of 1 ug/kg/day during gestation. Kidney defects are also common in the mouse, with embryotoxicity occurring as the dose is increased. The results of studies using strains of mice which are responsive and unresponsive to enzyme induction by 2,3,7,8-tetraCDD suggest that the occurrence of cleft palate may be controlled by the Ah gene locus. Responsive mice having high tissue concentrations of Ah receptor are more susceptible to this defect relative to unresponsive mice (U.S. EPA, 1985b).

In rats, teratogenicity produced by 2,3,7,8-tetraCDD is usually seen as subcutaneous edema, hemorrhage in the GI tract, and kidney malformation, which have been produced in animals dosed at levels greater than 0.1 ug/kg/day during gestation. There is evidence of enzyme induction in newborn rats after *in utero* exposure and through exposure during nursing (U.S. EPA, 1985b).

A three-generation reproduction study in the rat found adverse effects at doses greater than 0.001 ug/kg/day, which was considered a NOAEL. Doses of 0.01 and 0.1 ug/kg/day in the diet adversely affected reproduction. The use of this data for human risk assessment has been questioned due to differences in the dose a human infant would receive in milk relative to a rat (CARB and CDHS, 1986).

Studies in monkeys have been limited; typically few animals have been used. Fetotoxicity has been demonstrated, but there are insufficient data to clearly define a teratogenic response (U.S. EPA, 1985b). Fetotoxicity was produced in the monkey with an oral dose of 1 ug/kg administered either as a single dose or in multiple doses between days 20 and 40 of gestation (McNulty, 1985). The estimated NOAEL for the monkey is 0.002 ug/kg/day (CARB and CDHS, 1986).

Rats treated with mixed isomers of hexaCDD during gestation showed an increase in the occurrence of cleft palate, edema and vertebral defects at 100 ug/kg/day. Fetotoxicity was produced at levels greater than 10 ug/kg/day, with a dose of 0.1 ug/kg/day producing no increase in fetal malformation (U.S. EPA, 1985b).

Human studies have mostly centered around the herbicides 2,4,5-T and chlorophenols containing 2,3,7,8-tetraCDD as a contaminant. Evidence from studies done to date is not sufficient to

characterize adverse effects on human reproduction (U.S. EPA, 1985b). Human exposure in the chemical industry, during the Vietnam War from Agent Orange, and in forestry operations has not been able to define a teratogenic or other adverse effect on reproduction related to 2,3,7,8-tetraCDD. The animal data conclusively demonstrate that 2,3,7,8-tetraCDD is teratogenic and fetotoxic at low levels of exposure, and indicate a need to better define the potential for adverse reproductive effects in humans (U.S. EPA, 1985b).

MUTAGENICITY

Early in vitro studies with 2,3,7,8-tetraCDD produced positive results in test systems without mammalian metabolic activation in Salmonella typhimurium strain TA 1532, which is useful for detecting mutagens causing frameshift mutations. In these studies there was no indication of mutagenicity in strains used for detecting point mutations, such as strain TA 1530 (CARB and CDHS, 1986).

Later in vitro studies using the same or similar strains of S. typhimurium with or without mammalian metabolic activation have produced negative results. A positive mutagenic response has been seen in vitro with test cells such as E. coli SD-4, S. cervisiae and cultured mouse lymphoma cells (CARB and CDHS, 1986). Many of these studies are difficult to interpret due to solubility problems with 2,3,7,8-tetraCDD, poor cell survival or solvent related effects on the test cells. The capacity for 2,3,7,8-tetraCDD to produce mutation seems to be low, but remains questionable (Kociba, 1984).

In vivo studies in mice and rats designed to detect chromosome abnormalities have produced conflicting results. Studies in humans exposed to 2,3,7,8-tetraCDD either occupationally or accidentally have also produced questionable data and are insufficient to evaluate the capacity of 2,3,7,8-tetraCDD to produce chromosome aberrations in man (U.S. EPA, 1985b).

Of the CDFs, 2,9-diCDF, 2,6-diCDF, 2,3,7,8-tetraCDF and octaCDF produced no evidence of mutagenicity in S. typhimurium strains TA 98 and TA 100. 2,3,7,8-TetraCDF also produced negative results in strains TA 1535, TA 1537 and TA 1978. A mixture of CDFs did produce a dose-related response for increasing sister chromatid exchange (SCE) in cultured chinese hamster lung cells (NRCC, 1984).

Evaluation of the mutagenicity of CDDs and CDFs, particularly 2,3,7,8-tetraCDD, is difficult due to conflicting results from existing studies. While 2,3,7,8-tetraCDD does show some indication of being weakly mutagenic, its high toxicity may provide only a small range of doses for a mutagenic response to be seen (U.S. EPA, 1985b). The U.S. EPA (1985b), IARC (1982),

and CARB and CDHS (1986) are all in agreement that there is not sufficient evidence to determine the genotoxicity of 2,3,7,8-tetraCDD to man.

CARCINOGENICITY

Animal Studies

Chronic studies in rats and mice have demonstrated the carcinogenicity of 2,3,7,8-tetraCDD. Table 4.5 summarizes the major studies available on the carcinogenicity of various CDDs.

A two year 2,3,7,8-tetraCDD feeding study using Sprague-Dawley rats was conducted by Kociba et al. (1978) at levels of 0.001, 0.01, and 0.1 ug/kg/day in the diet (U.S. EPA, 1985b). At the 0.01 and 0.1 ug/kg/day levels, a significant increase in hepatocellular carcinoma and hepatocellular neoplastic nodules was seen in both sexes. The 0.1 ug/kg/day level produced carcinoma of the hard palate and carcinoma of the nasal turbinates in both sexes, carcinoma of the lung in females, and carcinoma of the tongue in males. This study, determined by the U.S. EPA (1985b) to have been properly carried out, reported a significant increase in tumor response at the 0.01 and 0.1 ug/kg/day levels. The International Agency for Research on Cancer (IARC, 1982) and CARB and CDHS (1986) also consider this study adequate as a bioassay for carcinogenicity.

In a two year gavage study by NTP (1982a) using B6C3F1 mice, males were dosed with 2,3,7,8-tetraCDD at 0.01, 0.05, and 0.5 ug/kg/week and females at 0.04, 0.2, and 2.0 ug/kg/week. Males in the high dose group had a statistically significant increase in hepatocellular carcinoma. Females in the high dose group had a significant increase in hepatocellular adenoma and carcinoma, subcutaneous fibrosarcoma, thyroid follicular-cell adenoma and adrenal cortical adenoma. Where NTP considered an increase in histiocytic lymphoma to be dose-related, CARB and CDHS (1986) did not, based on the incidence of lymphoma in all control groups in the study.

A study, done by Toth et al. (1979) and summarized by U.S. EPA (1985b), used Swiss mice in a gavage study with 2,3,7,8-tetraCDD given weekly at 0.007, 0.7, and 7.0 ug/kg for a year. The mice were then followed over the course of a lifetime with pathology emphasizing liver neoplasm incidence. A significant increase in liver tumors was seen only in the 0.7 ug/kg/week treatment group. Although an increase in liver tumors was seen in the high dose group, it was not statistically significant but did appear to be dose related. Survival in the high dose group was poor, and may explain the lack of a significant tumor response. This study is considered by U.S. EPA (1985b) to provide only suggestive evidence of carcinogenicity.

TABLE 4.5

COMPARATIVE CARCINOGENICITY OF ORALLY ADMINISTERED CDDs

Dibenzo-p-dioxin	Strain/species	Dose	Response
Unsubstituted	Osborne-Mendel Rats	5,000 or 10,000 ppm	No carcinogenic response.
	B6C3F1 Mice	5,000 or 10,000 ppm	No carcinogenic response.
2,7-DiCDD	Osborne-Mendel Rats	5,000 or 10,000 ppm	No carcinogenic response.
	B6C3F1 Mice	5,000 or 10,000 ppm	No carcinogenic response in females, suggestive evidence in males.
2,3,7,8-TetraCDD	Sprague-Dawley Rats	0.1, 0.01 & 0.001 ug/kg/day	Significant increase in hepatocellular carcinomas and hyperplastic nodules in female rats at both the intermediate and high-dose levels. At the high-dose, there was a significant increase in carcinomas of the hard palate/nasal turbinates in both sexes, of the tongue in males and of the lungs in females.
	Osborne-Mendel Rats	0.5, 0.05 & 0.01 ug/kg/week	Statistically significant increase in hepatocellular carcinomas, subcutaneous fibrosarcomas and adrenal cortical adenomas in high-dose females. Significant increase of thyroid tumors in male rats at all dose levels.

TABLE 4.5 (continued)

COMPARATIVE CARCINOGENICITY OF ORALLY ADMINISTERED CDDs

Dibenzo-p-dioxin	Strain/species	Dose	Response
	B6C3F1 Mice	0.01, 0.05 & 0.5 ug/kg/week in males and 2.0, 0.2 & 0.04 for females	Statistically significant increase of hepatocellular carcinomas in the high-dose males and females, and thyroid tumors, subcutaneous fibrosarcomas and histiocytic lymphomas in females.
1,2,3,6,7,8 HexaCDD (31%) + 1,2,3,7,8,9 HexaCDD (67%) mixture	Osborne Mendel Rats	1.25, 2.5 & 5 ug/kg/week	In male rats, the liver tumor incidence was significantly increased over control values only in the high-dose groups, while in female rats the incidence was significantly greater at both the medium and high dose groups.
	B6C3F1 Mice	1.25, 2.5 & 5 ug/kg/week for males and 2.5, 5.0 & 10 ug/kg/week for females	Liver tumor incidence was significantly increased in both male and female mice in the high-dose groups compared to control values.

Table compiled from U. S. EPA, 1985b; NRCC, 1981; Poiger and Schlatter, 1983.

As discussed by U.S. EPA (1985b), Van Miller (1977a,b) administered 2,3,7,8-tetraCDD in the diet to Sprague-Dawley rats at levels ranging from 0.0003 to 500 ug/kg/week for a period of 78 weeks. Survival was decreased in groups treated at levels greater than 24 ug/kg/week. Tumors of the lung and neoplastic nodules in the liver were significantly increased in the 2 ug/kg/week treatment group. Tumors were not seen in either control or low dose animals, which is unexpected for this strain of rat. Both U.S. EPA (1985b) and CARB and CDHS (1986) consider this study to provide suggestive evidence only, and is inadequate to determine a carcinogenic effect.

A 2,3,7,8-tetraCDD dermal study in Swiss-Webster mice of both sexes by NTP (1982b) produced significant increase in integumentary system fibrosarcomas in female mice, but not in males.

A gavage study by NTP (1980a), as discussed by U.S. EPA (1985b), used Osborne-Mendel rats and B6C3F1 mice administered a mixture containing 31 percent 1,2,3,6,7,8-hexaCDD, 67 percent 1,2,3,7,8,9-hexaCDD, and other CDDs including 2,3,7,8-tetraCDD as impurities. Rats of both sexes and male mice received 1.25, 2.5, and 5 ug/kg/week, and female mice 2.5, 5.0, and 10 ug/kg/week. Female rats had statistically significant increases in liver neoplastic nodules at all dose levels. Mice of both sexes in the high dose groups had a significant increase in hepatocellular adenoma or carcinoma.

There has been some controversy with regard to certain aspects of this latter study, with questions being raised about both the histologic preparation, and the pathologic interpretation of tissues. The female rat liver tissue slides have been reevaluated by several independent pathologists, and in all cases fewer neoplastic nodules and carcinomas were determined than in the original NTP interpretation (EPA, 1985b). Even though fewer such lesions were actually present than originally reported, the incidence in the high-dose female group was still statistically significant, and a dose-related response was seen when all groups were considered (CARB and DHS, 1986). The contribution of 0.09 percent tetraCDD present in the test mixture was also determined to be of no significance to the observed liver tumor incidence attributed to this hexaCDD mixture (EPA, 1985b).

A dermal study in Swiss mice of both sexes, conducted by NTP, (1980b) with the same hexaCDD mixture produced no significant increase in tumors, as reported by U.S. EPA (1985b). A small increase in fibrosarcomas of the integumentary system was seen, but was not considered significant.

Human Case Studies and Epidemiology

Human exposure to CDDs and CDFs has usually been associated with the manufacture or use of chemical products which contain them as contaminants. CDDs and CDFs have been found in the low parts per million (ppm) level in phenoxyacid herbicides such as 2,4,5-trichlorophenoxyacetic acid (2,4,5-T), in PCBs (Bowes et al., 1975), and in chlorinated phenols such as the wood preservative pentachlorophenol (PCP).

During the Vietnam War, Agent Orange, a mixture of the herbicides 2,4-dichlorophenoxyacetic acid (2,4-D) and 2,4,5-T was used extensively as a defolient. 2,3,7,8-TetraCDD was found as a contaminant at low ppm levels. U.S. military personnel involved in the spraying and persons exposed on the ground have been the subject of epidemiological studies (Wolfe et al., 1985).

Industrial accidents in chemical manufacturing plants have exposed large numbers of people to chemical products containing CDDs and CDFs as contaminants. Exposed persons have included plant workers and people living in nearby neighborhoods. The incident in Seveso, Italy in 1976, resulting in the release of 2,4,5-trichlorophenol contaminated with 2,3,7,8-tetraCDD, is one of the better known accidents producing data on human exposure to CDD containing materials. Other accidents have also provided a basis for other epidemiological investigations.

Human consumption of rice oil contaminated with polychlorinated biphenyls (PCBs) and CDFs (Yusho poisoning) has provided data for epidemiological studies. In one case 1200 people in Japan during 1968 ingested this contaminated oil, which contained PCBs at levels of 1000 ppm. The PCBs contained CDFs at levels of 5 ppm, mostly tetra- and pentaCDFs, with 2,3,7,8-tetraCDF present at a relatively high level of 0.45 ppm (Huff et al., 1980; Bowes et al., 1978).

These studies of human exposures have been reviewed by the U.S. EPA (1985b), IARC (1982), and CARB and CDHS (1986) and evaluated for their statistical power to determine carcinogenicity to humans. Most of these studies are of case/control and cohort study types, and have several deficiencies which limit their usefulness (CARB and CDHS, 1986):

- Amounts of CDDs/CDFs can only be estimated, and the dose received cannot be defined quantitatively.
- All exposures have involved CDDs and CDFs in combination with other chemicals, if they were present at all. Human exposure to only CDDs and CDFs has not been studied.

- Persons have generally been exposed for only short periods of time relative to a human life time.

- Sample sizes under study have been small. The small numbers of subjects involved do not allow the detection of small increases in tumor occurrence.

The studies resulting from exposure situations such as these have produced both positive and non-positive results. The positive associations of carcinogenicity in humans have provided only what is considered limited, suggestive evidence that phenoxyacetic acid herbicides, chlorophenols, and their CDD and CDF contaminants are capable of causing cancer in man.

CARB and CDHS (1986) have summarized the major epidemiological studies in Tables 4.6 and 4.7, and have calculated the statistical power of each. Only a non-positive human study (no effect seen) which has the statistical power to detect a 50 percent increase in risk should be used as evidence of "no effect". A statistical power of 0.80 or greater is usually considered adequate to detect a small increase in risk. Such statistical power is achieved by studies with larger sample sizes which are followed over a longer period of time (CARB and CDHS, 1986). CARB and CDHS (1986) prefer that a study should be able to detect the increase in cases that would be predicted to occur using exposure values and risk estimates obtained from animal studies.

Carcinogenicity Summary

Both U.S. EPA (1985b) and CARB and CDHS (1986) agree with IARC (1982) that the evidence from studies using rats and mice is sufficient to classify 2,3,7,8-tetraCDD as a carcinogen in these species. While evidence from human exposures has provided some suggestive evidence of carcinogenicity, it is considered inadequate by U.S. EPA (1985b) and CARB and CDHS (1986) to determine the carcinogenicity of 2,3,7,8-tetraCDD to man. The U.S. EPA (1985b) considers mixtures of phenoxyacetic acid herbicides or chlorophenols and their 2,3,7,8-tetraCDD contaminant "probably" carcinogenic to man.

The mixture of 1,2,3,6,7,8- and 1,2,3,7,8,9-hexaCDDs is considered an animal carcinogen by both U.S. EPA (1985b) and CARB and CDHS (1986), based on data obtained from the rat and the mouse. This mixture is also considered a potential human carcinogen. There are presently insufficient data to definitively assess the carcinogenicity of other CDDs or CDFs.

TABLE 4.6

SUMMARY OF MAJOR CASE/CONTROL STUDIES OF CDD EXPOSURE
(CARB and CDHS, 1986)

NATURE OF EXPOSURE	NO. OF CASES	POTENTIAL LATENCY	RESULTS	IF NEGATIVE, POWER TO DETECT A 50% INCREASE IN RISK (ONE-SIDED 95% TEST)	REFERENCES
Self-reported history of using herbicides 1 day	52 Male cases of soft tissue sarcoma	≥ 5 yrs	Significant odds ratio for exposure to phenoxy acids and chlorophenols		Hardell and Sandstrom, 1979
Self-reported history of using herbicides > 1 day	110 Male cases of soft tissue sarcoma	≥ 5 yrs	Significant odds ratio for exposure to phenoxy acids and chlorophenols		Erickson et al., 1981
Self-reported history of using chemicals > 1 day	71 Male cases of naso-pharyngeal cancer	≥ 5 yrs	Significant odds ratio for exposure to chlorophenols (adjusting for wood working)		Hardell et al., 1982

TABLE 4.6 (continued)

SUMMARY OF MAJOR CASE/CONTROL STUDIES OF CDD EXPOSURE
(CARB and CDHS, 1986)

NATURE OF EXPOSURE	NO. OF CASES	POTENTIAL LATENCY	RESULTS	IF NEGATIVE, POWER TO DETECT A 50% INCREASE IN RISK (ONE-SIDED 95% TEST)	REFERENCES
Occupational data from cancer registry	834 Male cases of nasal, sinus, and naso-pharyngeal cancer	Not Given	No association with chlorophenol exposure	0.48	Olsen and Jensen, 1984
Self-reported history of using herbicides ≥ 5 days	82 Male cases of soft tissue sarcoma	≥ 10 yrs	No significant association with phenoxy herbicides	0.25	Smith et al., 1984
Self-reported history of Vietnam service or using herbicides	281 Male cases of soft tissue sarcoma	4-14 yrs	No association with exposure to Agent Orange, or 2,4,5-T	0.26	Greenwald et al., 1984

TABLE 4.7

SUMMARY OF MAJOR COHORT STUDIES OF CDD EXPOSURE
(FROM CARB AND CDHS, 1986)

NATURE OF EXPOSURE	NO. OF SUBJECTS	POTENTIAL LATENCY	RESULTS	IF NEGATIVE, POWER TO DETECT A 50% INCREASE IN RISK (ONE-SIDED 95% TEST)	REFERENCES
Trichlorophenol process accident	121 Males	29 yrs	No excess total cancer *SMR for lung cancer = 1.8 (not significant)	For Total Cancer = 0.38 For Lung Cancer = 0.14	Zack and Suskind, 1980
Employment in a trichlorophenol process	39 Males with "high exposure potential" 22 Males with "low exposure potential"	15 yrs	SMR for total cancer = 1.9 (not significant)	For Total Cancer = 0.10	Cook et al., 1980
Employment in a 2,4,5,-T plant	204 Males, but only 47 exposed > 1 yr	20 yrs	No excess total cancer	For Total Cancer = 0.18	Ott et al., 1980

* SMR = Standardized Mortality Ratio: Observed Deaths vs Expected Deaths

TABLE 4.7 (continued)

SUMMARY OF MAJOR COHORT STUDIES OF CDD EXPOSURE

NATURE OF EXPOSURE	NO. OF SUBJECTS	POTENTIAL LATENCY	RESULTS	IF NEGATIVE, POWER TO DETECT A 50% INCREASE IN RISK (ONE-SIDED 95% TEST)	REFERENCES
2,4,5-T process accident	74 Males	Mean = 23 yrs	*SMR for total cancer = 1.7 (not significant) SMR for stomach cancer = 4.3 (significant at at 95% one-sided level)	For Total Cancer = 0.17	Theiss et al., 1982
Herbicide process accident	141 Males	20 yrs	SMR for total cancers = 1.2 (not significant)	For Total Cancer = 0.34	Dalderup and Zellenrath, 1983
Herbicide applicators	1926 Males	≥ 10 yrs	No excess total cancer	For Total Cancer = 0.68	Riithimaki et al., 1983
Employment in a 2,4,5-T plant	884 Males, but number actually exposed to 2,4,5-T much less	Median Approx. 30 yrs	SMR for lung cancer = 1.4 SMR for bladder cancer = 9.9 (significant at 95% level)	For Total Cancer = 0.80 For Lung Cancer = 0.42	Zack and Gaffey, 1983

*SMR = Standardized Mortality Ratio: Observed Deaths vs Expected Deaths

Mechanism of Carcinogenicity

The mechanism by which the carcinogenic CDDs and CDFs induce a tumor response is presently not understood (see Figure 4.1). In addition to the known capacity of 2,3,7,8-tetraCDD to produce tumors in animals, studies have provided an indication of tumor initiation, promotion, co-carcinogenicity, and also inhibition of tumors initiated by other carcinogens.

Dermal studies in mice have produced both positive and negative results for tumor promotion by 2,3,7,8-tetraCDD of tumors initiated by 7,12-dimethylbenz(a)anthracene (DMBA). In the rat, 2,3,7,8-tetraCDD was a promoter of liver carcinogenesis initiated by diethylnitrosamine. The mixture of 1,2,3,6,7,8- and 1,2,3,7,8,9-hexaCDD was not a promoter of tumors initiated by DMBA in a dermal study with mice (U.S. EPA, 1985b).

A response suggestive of tumor initiation was seen when 2,3,7,8-tetraCDD was applied to the skin of mice as an initiator, with the tumor promoter 12-0-tetradecanoylphorbol-13-acetate (TPA) applied later. Co-carcinogenicity through enzyme induction is also suggested as a possibility (CARB and CDHS, 1986).

In mice, 2,3,7,8-tetraCDD has also inhibited the tumor response produced by DMBA when both are applied to the skin. In this study, the time of application of 2,3,7,8-tetraCDD relative to DMBA was important to the outcome. The tumor response was inhibited to the greatest extent if 2,3,7,8-tetraCDD was applied 1 to 5 days before the DMBA, with less inhibition produced if 2,3,7,8-tetraCDD was applied 10 days before the DMBA. No decrease in tumor response was seen if 2,3,7,8-tetraCDD was applied just before or five days after initiation with DMBA. Similar results were obtained when 3-MC and benzo(a)pyrene (BaP) were used as initiators. In contrast, BaP-diol-epoxide tumor response was decreased to a greater extent when the 2,3,7,8-tetraCDD was applied either three days or five minutes before, or one day after BaP-diol-epoxide, implying that different mechanisms of tumor inhibition may be involved (U.S. EPA, 1985b).

SUMMARY AND DISCUSSION

The importance of molecular structure, specifically the number and location of the chlorine atoms on the CDD or CDF molecule, has proved to be an essential consideration when comparing the toxicity of individual congeners of both classes. Those congeners chlorinated in the 2,3,7 and 8 positions having a total of four, five, six or seven chlorine atoms possess the greatest toxicity, with 2,3,7,8-tetraCDD the most toxic of any CDD or CDF, and 2,3,7,8-tetraCDF the most toxic CDF. This relationship between molecular structure and toxicity is apparent for a number

of compound-related effects, and becomes important when evaluating the potential toxicity of mixtures containing multiple CDD and CDF congeners. The occurrence of various congeners in combination with one another is more the rule than the exception when dealing with environmental contamination.

Absorption from the GI tract and distribution to tissues also varies between species. The vehicle used to administer the dose affects absorption, and in cases where 2,3,7,8-tetraCDD is administered in combination with soil particles, bioavailability is decreased and less of the dose is absorbed. The effect soil (or other matrices) has on altering the bioavailability of CDDs and CDFs must be considered in any risk assessment, especially if a site-specific approach is to be used. In general, the liver and tissues with high lipid content sequester a greater part of the dose.

After metabolism, which occurs primarily in liver by cytochrome P_1-450, most 2,3,7,8-tetraCDD is excreted in urine or bile, with the biological half-life varying between species. The number and location of chlorine atoms on the CDD or CDF molecule produces differences in the half-life, and isomers within an isomer group may be metabolized and excreted at different rates. CDDs and CDFs have rather long retention times in tissues such as adipose, with a half-life of about a year for the monkey and 5.8 years for man.

There are considerable qualitative and quantitative differences between species in sensitivity to the toxic effects of CDDs and CDFs. Some effects, such as adverse effects on the thymus gland and the liver, are seen in nearly all species tested, while others such as skin lesions are not. Acute toxicity is a good example of a quantitative difference in toxicity between species, with an LD_{50} of 0.6 ug/kg for the male guinea pig, and 5,051 ug/kg for the male hamster. As indicated below, this very low LD_{50} shows 2,3,7,8-tetraCDD to be one of the most acutely toxic substances known (Doull et al., 1980):

APPROXIMATE ACUTE LD_{50}s FOR
SELECTED HIGHLY TOXIC SUBSTANCES

Agent	LD_{50} (ug/kg)
Botulinus Toxin	0.01
2,3,7,8-TetraCDD	0.6
Tetrodotoxin	100
Aldicarb	800
Strychnine sulfate	2000

Within a species there are also quantitative differences in toxicity between isomers of an isomer group, with LD_{50}s in the guinea pig of 3.1 ug/kg for the 1,2,3,7,8-pentaCDD isomer, and .e

1,125 ug/kg for the 1,2,4,7,8-pentaCDD isomer. Even after acute exposures death is prolonged, with a decrease in bodyweight known as "wasting" occurring over time in most animals tested. In man chloracne is the most common effect after exposure. Other adverse effects seen after human exposure include liver abnormalities, hematologic disorders, hyperpigmentation and neurological changes. Such effects may be of short duration or may persist for years.

A number of toxic effects produced by 2,3,7,8-tetraCDD and related compounds are believed to be mediated through binding to a receptor protein designated as the Ah receptor, which has been found in cells of a number of tissues in animals, and to some extent in humans. For certain effects including P_1-450 enzyme induction and aryl hydrocarbon hydroxylase (AHH) activity, there is a correlation between binding of the CDD or CDF molecule to this receptor and biochemical effect in a number of species. The potency of a CDD or CDF congener for producing such receptor-mediated effects is related to the number and location of the chlorine atoms on the molecule, and is a structure-activity related phenomenon. 2,3,7,8-TetraCDD is the most potent CDD or CDF, and 2,3,7,8-tetraCDF the most potent CDF producing these effects. For other effects there is some correlation between the presence of this receptor and toxicity for a few species, which is not seen in most species tested. What role, if any, the Ah receptor has in the carcinogenicity of CDDs and CDFs has not been determined.

Tetratogenicity and other adverse effects on reproduction, including fetotoxicity, have been produced in several animal species at very low levels of exposure. Cleft palate is characteristic in the mouse, with kidney defects also frequent after exposure to 2,3,7,8-tetraCDD. There is some evidence which indicates that the cleft palate defect may be genetically related to the Ah locus and induction of AHH activity, which is mediated by 2,3,7,8-tetraCDD binding to the Ah receptor. Fetotoxicity has been seen in the monkey and in the rat. Studies in humans resulting from environmental or occupational exposures have not provided sufficient data to determine either teratogenicity or other adverse effects on reproduction. Such data are also inadequate to eliminate the possibility that such effects would occur in humans.

Studies on the genotoxicity of CDDs and CDFs have produced conflicting or otherwise questionable results in most *in vitro* and *in vivo* test systems, and are considered inadequate to determine the genotoxicity of 2,3,7,8-tetraCDD. 2,3,7,8-TetraCDD and a mixture of two hexaCDD isomers have been determined to be carcinogens in the mouse and the rat. Neoplasms of the liver are most commonly seen, with tumors of the lung and thyroid gland also occurring. Other CDD or CDF congeners chlorinated in the 2,3,7, and 8 positions and having a total of four, five, six or

seven chlorine atoms are also of concern in the absence of valid carcinogenicity bioassays. There has been some controversy about whether 2,3,7,8-tetraCDD should be classified as a tumor initiator or as a tumor promotor. The CDHS (CARB and CDHS, 1986) considers any carcinogen to have a non-threshold mechanism of action, and unless evidence is sufficient to determine that the mechanism of action has a threshold, it does not distinguish between an initiator or a promoter. The CDHS also does not believe that toxic potency values derived from structure-activity relationships, along with acute, subchronic and in vitro studies are adequate to estimate carcinogenic potency (See Appendix E).

The U.S. EPA, in the Chlorinated Dioxins Workgroup Position Document of April 1985, has determined that as an interim measure, the toxic risks of complex mixtures can be reasonably estimated by considering the distribution of those CDDs and CDFs chlorinated in the 2,3,7, and 8 positions. The Scientific Advisory Board's Dioxin Toxic Equivalency Methodology Subcommittee evaluated the EPA method in a November 1986 report, and concluded that the method is a reasonable interim means of assessing the risk presented by exposure to complex mixtures of CDDs and CDFs, at least until the method has been validated by testing, or until better data become available on more congeners. Both the EPA and the CDHS approaches are discussed in greater detail in Chapter 6 of this report.

Human studies resulting from environmental or occupational exposure to phenoxyacetic acid herbicides, such as Agent Orange in Vietnam, and chlorophenol products containing CDDs or CDFs as contaminants have provided human epidemiological data. Ingestion of rice oil contaminated with CDFs (Yusho poisoning) in separate incidents in Taiwan and Japan have also formed the basis for additional human studies. Due to limitations involving uncertainty of the dose received, the presence of other agents, length of exposure, and generally small sample sizes, these studies are inadequate to determine the carcinogenicity of CDDs and CDFs to man.

Based on animal data, 2,3,7,8-tetraCDD and a mixture of 1,2,3,6,7,8- and 1,2,3,7,8,9-hexaCDDs are considered potential human carcinogens. The U.S. EPA (1985b) Carcinogen Assessment Group has calculated a "relative potency index" for comparing the carcinogenic potency of 55 suspect human carcinogens. 2,3,7,8-TetraCDD was the most potent carcinogen ranked, and is compared below with the next two ranked chemicals and TCE, a common ground water contaminant in California:

RELATIVE CARCINOGENIC POTENCY

Chemical	Potency Relative to 2,3,7,8-TetraCDD
2,3,7,8-TetraCDD	1
1,2,3,6,7,8-/1,2,3,7,8,9-HexaCDD mixture (31%/67%)	1/20
Bis(chloromethyl) ether	1/50
Trichloroethylene (TCE)	1/50,000,000

Data are not available to assess the carcinogenicity of other CDDs or CDFs.

CHAPTER 5. CRITERIA, STANDARDS, AND REGULATIONS

CRITERIA AND STANDARDS

Available criteria and standards for CDDs in water and other media are shown in Table 5.1. These only address 2,3,7,8-tetraCDD and the hexaCDDs: criteria have not been recommended for the CDFs and the other CDDs. The values listed in Table 5.1 are based upon different assumptions and have differing applications depending on their derivation. The CDD criteria and standards were developed based upon extrapolations from animal laboratory or human epidemiology studies of acute and chronic effects of CDDs. Where information was available, an explanation of the derivation of the concentration limit, as well as a description of how the limit should be applied is presented.

The criteria for 2,3,7,8-tetraCDD are generally based on dose-response data from animal studies. In many of the derived criteria, the linearized multi-stage model is used for low dose extrapolation. This model assumes that there is no threshold in the dose response curve. The model yields estimates of risk that are conservative, representing an upper limit (95 percent upper confidence limit) for the risk. In other words, it is unlikely that the actual risk is higher than the risk predicted by this model. The derived criterion is set equal to the upper 95 percent confidence limit for one excess lifetime cancer per one million people. This approach is used by the U. S. Environmental Protection Agency (U.S. EPA), California Department of Health Services (CDHS), Center for Disease Control (CDC), and the California Air Resources Board (CARB) in deriving their criteria for carcinogens.

Where adverse effects (other than cancer) are used to derive a criterion, uncertainty factors are applied. In these situations, the dose, expressed in milligrams of chemical per kilogram (mg/kg) of body weight, is divided by an uncertainty (or safety) factor to obtain an Acceptable Daily Intake (ADI) or a Suggested No Adverse Effect Level (SNARL). The uncertainty factor generally ranges from 10 to 1,000 and reflects the quality of the toxicological data, the degree of confidence in the data and the nature of the effects of concern (the more secure the data base, the lower the uncertainty factor). In contrast to the no-threshold approach used to determine risks of a carcinogen, the ADI and SNARL assume a threshold below which adverse effects do not occur. This approach was used by the U.S. Food and Drug Administration (FDA), the National Academy of Science (NAS), Ontario (Canada) Ministry of the Environment, National Research Council of Canada, and New York State in deriving criteria or setting standards for CDDs.

TABLE 5.1
CRITERIA AND STANDARDS

COMPOUND	AGENCY/ APPLICATION	CRITERION[1/]	NOTES	REFERENCE
2,3,7,8-tetraCDD	U. S. FDA (Advisory for Great Lakes States)	.025 ug/kg (25 ppt) in fish flesh	Fish flesh containing greater than or equal to .025 ug/kg, but less than .050 ug/kg should not be consumed more than twice a month.	U.S. FDA, 1983
"	"	.050 ug/kg (50 ppt) in fish flesh	Fish flesh containing greater than or equal to .050 ug/kg should not be consumed. FDA premises its exposure assessment on the assumption that only limited amounts of fish having 2,3,7,8-tetra CDD concentrations at or near the advisory level will actually be consumed. FDA's estimate of the carcinogenic potential of 2,3,7,8-tetraCDD is 1.75×10^{4} cancers per mg/kg/day. This is one ninth the potency calculated by U.S. EPA for determination of Ambient Water Quality Criteria.	"
2,3,7,8-tetraCDD	U.S. EPA (Ambient Water Quality Criteria)	1.3×10^{-8} ug/l in ambient water (one in one million excess lifetime cancer risk)	This criterion is based upon daily ingestion of 2 liters of drinking water and consumption of 6.5 grams of fish and shellfish. U.S. EPA assumes that approximately 94% of the 2,3,7,8-tetraCDD exposure results from the consumption of aquatic organisms which exhibit an average bioconcentration potential of 5,000 fold.	U.S. EPA, 1984a
"	U.S. EPA (Carcinogen Assessment Group Level)	1.4×10^{-8} ug/l in ambient water (one in one million excess lifetime cancer risk)	This criterion is based upon ingestion of fish and shellfish only. U.S. EPA's estimate of the carcinogenic potential of 2,3,7,8-tetraCDD is 1.5×10^{5} cancers per mg/kg/day. This is nine times the potency calculated by FDA for determination of action levels.	"

COMPOUND	AGENCY/ APPLICATION	CRITERION[1/]	NOTES	REFERENCE
(continued)	"	2.2×10^{-7} ug/l ambient water (one in one million excess lifetime cancer risk)	This criterion is based upon drinking 2 liters of water per day.	"
2,3,7,8-tetraCDD	U.S. EPA (Carcinogen Assessment Group)	6.4×10^{-9} ug/kg/day total intake from all sources in humans	This criterion represents the intake which should not be exceeded in order to keep the excess cancer cases below one in one million people. It is based upon the upper limit incremental cancer risk of 1.56×10^{-1} per ng/kg/day of 2,3,7,8-tetraCDD, converted to a concentration as follows: $\frac{(1 \times 10^{-3} \text{ ug/kg/day}) \times (1 \times 10^{-6})}{1.56 \times 10^{-1}} =$ 6.4×10^{-9} ug/kg/day	U.S. EPA, 1985b
"	"	2.2×10^{-7} ug/l in drinking water	This criterion represents the concentration which should not be exceeded in order to keep the excess cancer cases below one in one million people. It is based upon the upper limit estimate of the incremental cancer risk of 4.5×10^{-3} for a continuous lifetime exposure to .001 ug/l of 2,3,7,8-tetraCDD in drinking water, converted to a concentration as follows: $\frac{.001 \text{ ug/l} \times (1 \times 10^{-6})}{4.5 \times 10^{-3}} =$ 2.2×10^{-7} ug/l	"

COMPOUND	AGENCY/ APPLICATION	CRITERION[1/]	NOTES	REFERENCE
(continued)	"	3.0×10^{-8} ug/m^3 in ambient air	This criterion represents the concentration which should not be exceeded in order to keep the excess cancer cases below one in one million people. It is based upon the upper limit inhalation risk estimate of 3.3×10^{-5} for a continuous lifetime exposure to 1×10^{-6} ug/m^3 of 2,3,7,8-tetraCDD in ambient air. $\frac{(1 \times 10^{-6} \text{ ug/m}^3) \times (1 \times 10^{-6})}{3.3 \times 10^{-5}} =$ 3.0×10^{-8} ug/m^3	U.S. EPA, 1987b
2,3,7,8-tetraCDD	NAS (Suggested No Adverse Response Level)	7×10^{-4} ug/l in drinking water	This advisory level assumes a threshold effect for 2,3,7,8-tetraCDD. The threshold is a level below which no adverse effects are believed to occur. It assumes that an average 70 kg human adult consumes 2 liters of drinking water per day and that 20% of total intake is from water. It incorporates the highest No Observed Adverse Effect Level (NOAEL) of .01 ug/kg/day from rat studies and an uncertainty factor of 100 to derive an Allowable Daily Intake (ADI). $\text{ADI} = \frac{0.01 \text{ ug/kg/day}}{100 \text{ (uncertainty factor)}} = 0.0001 \text{ ug/kg/day}$ $\text{SNARL} = \frac{0.0001 \text{ ug/kg/day} \times 70 \text{ kg} \times 20\%}{2 \text{ liters/day}} =$ 7×10^{-4} ug/l	NAS, 1977

TABLE 5.1 (continued)
CRITERIA AND STANDARDS

COMPOUND	AGENCY/ APPLICATION	CRITERION[1/]	NOTES	REFERENCE
2,3,7,8-tetraCDD	Ontario Ministry of the Environment (Ontario, Canada) (Criterion)	20 ng/kg (ppt) in fish flesh	This agency assumes that 2,3,7,8-tetraCDD is a threshold pollutant below which tumor production due to exposure of this compound would be unlikely. A NOAEL of .001 ug/kg/day and a safety factor of 100 yields the maximum ADI of 1×10^{-5} ug/kg/day for humans.	Ontario, Canada, 1986
2,3,7,8-tetraCDD	New York State (standard)	1×10^{-6} ug/l in ambient water	This standard is designed to prevent aquatic food tainting and is deemed to be protective of the health of human or wildlife consumers of fish and shellfish flesh for 2,3,7,8-tetraCDD.	New York, 1985
2,3,7,8-tetraCDD	New York State (standard)	3.5×10^{-5} ug/l in groundwater	This standard is designed to protect human health from 2,3,7,8-tetraCDD contaminated drinking water.	Zambrano, 1987
2,3,7,8-tetraCDD	New York State (criterion)	10 ng/kg (ppt) in fish flesh	This criterion is deemed to be protective of humans consuming fish.	Zambrano, 1987
2,3,7,8-tetraCDD	Michigan Department of Public Health (Advisory)	10 ppt in fish	This criterion is deemed to be protective of humans consuming fish. The tetraCDD dose is related to 1×10^{-5} risk for a 70 kg individual exposed for a lifetime.	Michigan, 1986
2,3,7,8-tetraCDD	Centers for Disease Control (Action Levels)	1 ppb in soil	This is an action level to protect against human exposure to contaminated soil. It was derived as a site specific value for a residential area in Missouri.	Kimbrough et al., 1984

TABLE 5.1 (continued)
CRITERIA AND STANDARDS

COMPOUND	AGENCY/ APPLICATION	CRITERION[1/]	NOTES	REFERENCE
2,3,7,8-tetraCDD	California Air Resources Board and Department of Health Servces (Toxic Air Contaminant Level)	3.0×10^{-8} ug/m^3 in ambient air	This proposed criterion is based upon the CDHS no-threshold, multi-stage model. It is a concentration in air which should not be exceeded in order to keep the excess cancer cases below one in one million people.	CARB and CDHS, 1986
2,3,7,8-tetraCDD	World Health Organization International Agency for Research on Cancer (Classification Status of 2,3,7,8-tetra CDD)	Level has not yet been developed.	This agency has classified 2,3,7,8-tetraCDD as a 2B chemical which means that there is sufficient animal evidence to indicate that it is a carcinogen; however, there is inadequate human evidence for carcinogenicity.	IARC, 1982
hexa-CDD	U.S. EPA (Carcinogen Assessment Group)	1.6×10^{-7} ug/kg/day from all sources in humans (ADI)	This criterion represents the concentration which should not be exceeded in order to keep the excess cancer cases below one in one million people. It is based upon the upper limit incremental cancer risk of 6.2×10^{-3} per ng/kg/day of hexaCDD converted to a concentration level as follows: $\frac{(1 \times 10^{-3} \text{ ug/kg/day}) \times (1 \times 10^{-6})}{6.2 \times 10^{-3}} = 1.6 \times 10^{-7}$ ug/kg/day	U.S. EPA, 1985b
"	"	5.5×10^{-6} ug/l in drinking water	This level represents the concentration which should not be exceeded in order to keep the excess cancer cases below one in one million people. It is based upon the upper limit estimate of the incremental cancer risk of 1.8×10^{-4} for a continuous lifetime exposure to .001 ug/l of hexa-CDD (isomer unspecified) in drinking water, converted to a concentration level as follows: $\frac{.001 \text{ ug/l} \times (1 \times 10^{-6})}{1.8 \times 10^{-4}} = 5.5 \times 10^{-6}$ ug/l	"

TABLE 5.1 (continued)
CRITERIA AND STANDARDS

COMPOUND	AGENCY/ APPLICATION	CRITERION[1/]	NOTES	REFERENCE
hexa-CDD	"	7.7×10^{-7} ug/m^3 in ambient air	This criterion represents the concentration which should not be exceeded in order to keep the excess cancer cases below one in one million people. It is based upon the upper limit of air inhalation risk estimate of 1.3×10^{-6} for a continuous lifetime exposure to 1×10^{-6} ug/m^3 of hexa-CDD in ambient air. $\frac{(1 \times 10^{-6} \text{ ug/m}^3) \times (1 \times 10^{-6})}{1.3 \times 10^{-6}} =$ 7.7×10^{-7} ug/m^3	U.S. EPA, 1985b
hexa-CDD	California Air Resources Board and Department of Health Services Toxic Air Contaminant Level	1.0×10^{-6} ug/m^3 in ambient air	This proposed level is based upon the CDHS no threshold, multi-stage model. It is a concentration level in air which should not be exceeded in order to keep the excess cancer cases below one in one million people.	CARB and CDHS, 1986
hexa-CDD	Southern California Edison Co. (Proposal for Guideline)	6.2×10^{-4} ug/l in drinking water	This proposed guideline is based upon the lowest dose tested by the National Cancer Institute and includes a safety factor of 10,000. It is a threshold guideline.	Jaegar, 1984
hexa-CDD	National Research Council of Canada (Criteria)	1.26×10^{-2} ug/l in ambient water for human consumption of fish	This criterion is based upon the Lowest Observed Effect Level (LOEL) of .36 ug/kg/day with a safety factor of 1,000.	NRCC, 1981
	"	2×10^{-2} ug/kg (ppb) in fish flesh	This criterion is the limit allowed in the flesh of Lake Ontario commercial fish exported to the United States.	"

1/ Or standard where noted.

Of the criteria and standards presented in Table 5.1, only the New York State standard is legally enforceable, while the remaining values are advisory. States and local agencies may adopt advisory levels as enforceable standards, or they may be used as the toxicological basis for considering control technology.

REGULATIONS

The regulations presented in Table 5.2 are generally related to waste disposal and effluent discharges of CDD and CDF impurities found in such products as pentachlorophenol and pesticides. Federal regulations have been promulgated under the Resource Recovery and Conservation Act (RCRA), the Clean Water Act (CWA), and the Federal Insecticide, Fungicide and Rodenticide Act (FIFRA).

SUMMARY AND DISCUSSION

There are no enforceable federal or California state standards for CDDs or CDFs. The advisory levels listed in Table 5.1 are guidelines only, with the exception of standards set by the state of New York.

The federal, state and provincial regulatory agencies which have proposed advisories or standards to protect humans consuming fish contaminated with 2,3,7,8-tetraCDD or hexaCDD are as follows:

Agency	Compound	Guideline
FDA	2,3,7,8-tetraCDD	≥25 ppt (fish flesh) Consume no more than twice per month ≥50 ppt (fish flesh) Unfit for human consumption
EPA (AWQC)	2,3,7,8-tetraCDD	1.4×10^{-8} ug/l (ingestion of fish and shellfish only)
EPA (AWQC)	2,3,7,8-tetraCDD	1.3×10^{-8} ug/l (ingestion of 2 liters drinking water and 6.5 grams of fish and shellfish)
EPA (CAG)	2,3,7,8-tetraCDD	6.4×10^{-9} ug/kg/day (intake from all sources)

Agency	Compound	Guideline
Ontario Ministry of the Environment	2,3,7,8-tetraCDD	20 ppt (fish flesh)
State of New York	2,3,7,8-tetraCDD	1×10^{-6} ug/l[1/] (for human and wildlife consumers of fish and shellfish)
State of New York	2,3,7,8-tetraCDD	10 ng/kg (ppt) (fish flesh)
State of Michigan	2,3,7,8-tetraCDD	10 ppt fish flesh (for humans consuming fish)
National Research Council of Canada (Ottawa)	hexaCDD	1.26×10^{-2} ug/l (for humans consuming fish)

1/ Standard
AWQC = Ambient Water Quality Criteria
CAG = Carcinogen Assessment Group

Some agencies have proposed advisories for drinking water as follows:

Agency	Compound	Guideline
EPA (AWQC)	2,3,7,8-tetraCDD	2.2×10^{-7} ug/l
EPA (CAG)	2,3,7,8-tetraCDD	2.2×10^{-7} ug/l
National Academy of Sciences	2,3,7,8-tetraCDD	7.0×10^{-4} ug/l
State of New York	2,3,7,8-tetraCDD	3.5×10^{-5} ug/l (groundwater)
EPA (CAG)	hexaCDD	5.5×10^{-6} ug/l

AWQC = Ambient Water Quality Criteria
CAG = Carcinogen Assessment Group

TABLE 5.2

REGULATIONS

COMPOUND	AGENCY	APPLICABLE REGULATIONS	REFERENCE
2,3,7,8-tetraCDD	CDHS	Soluble Threshold Limit Concentration (STLC) for 2,3,7,8-tetraCDD is .001 mg/1. (The STLC is based upon the potential for soluble substances from improperly disposed wastes to migrate via surface or ground water to sensitive aquifer systems such as drinking water supplies or aquatic wildlife environments.) Total Threshold Limit Concentration (TTLC) for 2,3,7,8 tetraCDD is 0.01 mg/1. (The TTLC is based upon the potential impacts on land, resulting from improper disposal of particulate toxic wastes.) STLCs and TTLCs are used to classify wastes as either hazardous or extremely hazardous. The method of disposal depends upon this classification of the wastes.	CAC, 1984
CDDs and CDFs	U.S. EPA	A final rule has been made which promulgates CDD and CDF treatment standards and prohibits land disposal of certain CDD- and CDF-containing wastes unless the treatment standards are achieved. Treatment standards for hexaCDDs, hexaCDFs, pentaCDDs, pentaCDFs, tetraCDDs, and tetraCDFs require that the waste extract be below the 1 ppb limit. Treatment standards for 2,4,5-trichlorophenol, 2,4,6-trichlorophenol, 2,3,4,6-tetrachlorophenol and pentachlorophenol require that the waste extract be below 50, 50, 100 and 10 ppb respectively. However, EPA has granted a nationwide two-year variance to the effective date (November 8, 1986), due to lack of alternative destructive technologies. During this two-year variance, wastes must be managed in facilities that are in compliance with Section 3004(o) [42 U.S.C. 6924(o)]. Citation: U.S. Environmental Protection Agency. November 7, 1986. Hazardous Waste Management System; Land Disposal Restrictions; Final Rule, Federal Register, 40 CFR Part 260, Vol. 51, No. 216. Washington, D.C.	U.S. EPA, 1986b

TABLE 5.2 (continued)

REGULATIONS

COMPOUND	AGENCY	APPLICABLE REGULATIONS	REFERENCE
CDDs and CDFs	U.S. EPA	Certain associated wastes from chlorophenolic formulations used by wood preserving or surface protection facilities (either at sawmills or at wood treaters before air seasoning) may be subject to regulation as acutely hazardous wastes under the Resource Conservation and Recovery Act (RCRA) because of their contamination with CDDs and CDFs. The hazardous waste listings which may apply are found in 40 CFR Part 260 et al. of the Code of Federal Regulations as Hazardous Wastes Nos. F020, F021, F022, F023, F026, F027, and F028. An explanation of these wastes follows: F020-Wastes (except wastewater and spent carbon from hydrogen chloride purification) from the production or manufacturing use (as a reactant, chemical intermediate, or component in a formulating process) of tri- or tetrachlorophenol or of intermediates used to produce their pesticide derivatives. (This listing does not include wastes from production of hexachlorophene from highly purified 2,3,5-trichlorophenol). F021-Wastes (except wastewater and spent carbon from hydrogen chloride purification) from the production or manufacturing use (as a reactant, chemical intermediate, or component in a formulating process) of pentachlorophenol, or of intermediates used to produce its derivatives. F022-Wastes (except wastewater and spent carbon from hydrogen chloride purification) from the manufacturing use (as a reactant, chemical intermediate, or component in a formulation process) of tetra-, penta-, or hexachlorobenzenes under alkaline conditions. F023-Wastes (except wastewater and spent carbon from hydrogen chloride (purification)) from the production of materials on equipment previously used for the production or manufacturing use (as a reactant, chemical intermediate, or component in formulating process) of tri- and tetrachlorophenols. (This listing does not include wastes from equipment used only for the production or use of hexachlorophene made from highly purified 2,4,5-trichlorophenol.)	U.S. EPA, 1985a

TABLE 5.2 (continued)

REGULATIONS

COMPOUND	AGENCY	APPLICABLE REGULATIONS	REFERENCE
CDDs and CDFs (cont.)	U.S. EPA	F026-Wastes (except wastewater and spent carbon from hydrogen chloride purification) from the production of materials or equipment previously used for the manufacturing use (as a reactant, chemical intermediate, or component in a formulation process) of tetra-, penta-, or hexachlorobenzenes under alkaline conditions. F027-Discarded unused formulations containing tri-, tetra-, or pentachlorophenols, or compounds from these chlorophenols. (This listing does not include formulations containing hexachlorophene synthesized from prepurified 2,3,5,-trichlorophenol as the sole component). F028-Residues resulting from incineration or thermal treatment of soil contaminated with U.S. EPA Hazardous Wastes Nos. F020, F021, F022, F023, F026, and F027.	
Pentachlorophenol	U.S. EPA	Process wastewater effluent discharges from wood preserving facilities which use arsenicals, chromates, creosote, and/or pentachlorophenol are regulated under the Clean Water Act (CWA). The final regulations were promulgated in 1981 and vary according to whether the facility was in existence at the time of the regulation (pretreatment standards for existing sources) or is a new plant (new sources performance standards). The release of pentachlorophenol and creosote in wood treatment wastewaters is controlled by the use of the indicator pollutant, oil and grease.	U.S. EPA, 1981

TABLE 5.2 (continued)

REGULATIONS

COMPOUND	AGENCY	APPLICABLE REGULATIONS	REFERENCE
Pentachloro-phenol	U.S. EPA	At present, wastewater treatment sludges from wood preserving processes which use creosote and/or pentachlorophenol are regulated as Hazardous Waste No. K001 under 40 CFR Part 261.31. This includes oil/water separator sludges at the bottom of surface impoundments used to treat or dispose of wastewater (percolation or evaporation ponds), filter media (carbon, sand, soil), spray irrigation fields (considered land treatment units), sludge dewatering/drying beds, etc. The RCRA management standards would not apply to top wastewater treatment sludges (or wastewaters if they are listed as hazardous wastes in the future) while they are managed on-site in tanks which meet certain design requirements of 40 CFR Part 264.1(g) (6) and Part 265.1 (c) (10). However, if sludges are removed from these units the full RCRA permitting requirements apply.	U.S. EPA, 1983
2,4,5-Trichloro-phenoxy acetic acid (2,4,5-T)	U.S. EPA	Because formulations of 2,4,5-T have been found to contain 2,3,7,8-tetra CDD as a contaminant, cancellations of registrations of products which contain 2,4,5-T as an active ingredient is in effect. Except for those products whose registrations were suspended in 1979, all existing stocks which were packaged and labeled for non-suspended end use(s) and released for shipment before the receipt of the October 18, 1983 Federal Register notice may be distributed and sold for one year after the effective date of cancellation. Under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) an individual or organization which is adversely affected by a cancellation may contest such action in an adjudicatory hearing. Because some registrants of 2,4,5-T products have pursued this course, regulatory action on this products will not be final until cancellation disputes have been resolved.	U.S. Congress, 1983

TABLE 5.2 (continued)

REGULATIONS

COMPOUND	AGENCY	APPLICABLE REGULATIONS	REFERENCE
Penntachloro-phenol (based on hexaCDD)	U.S. EPA	This regulation requires registrants of pentachlorophenol to reduce the concentration of hexaCDD in three phases: 1. The maximum batch concentration initially is 15 ppm. 2. After February 2, 1988, the maximum batch hexaCDD concentration will be 6 ppm, with a maximum monthly average of 3 ppm. 3. Finally, after February 2, 1989, the maximum batch hexaCDD concentration will be 4 ppm, with a maximum average of 2 ppm.	U.S. EPA, 1987

In addition to the above advisories and standards, the Centers for Disease Control in Atlanta, Georgia have recommended a cleanup level to EPA of 1 ppb 2,3,7,8-tetraCDD in soil, a site specific value for a residential area in Times Beach, Missouri.

Some state and federal regulations exist which control CDD and CDF impurities in PCP and pesticide compounds. These regulations put restrictions on pesticide registrations, waste and effluents, and specify treatment of material with CDD and CDF impurities (Table 5.2).

CHAPTER 6: WOOD TREATMENT PRACTICES AND CALIFORNIA SITE CONTAMINATION

This chapter presents a brief overview of wood treatment practices. Three California wood treatment facilities are described to provide examples of chlorophenol-related CDD and CDF contamination.

WOOD TREATMENT PRACTICES

Chlorophenols, such as pentachlorophenol (PCP), tetrachlorophenol and their potassium and sodium salts, creosote, coal tars and copper arsenate compounds have been used routinely at sawmills and wood treatment facilities in California. At sawmills, salts of PCP are used to treat freshly cut wood to prevent sap stains caused by the action of fungi and molds, an example being "blue stain" which leaves a dark discoloration on unprotected wood. Unlike pentachlorophenol, which is highly insoluble in water, the sodium and potassium salts (pentachlorophenates) are very soluble.

PCP is commonly sold as a solid containing 95 percent PCP, and is applied to wood products as a 5 percent solution after being dissolved in a petroleum solvent (Baker and Matheson, 1981). It is used also in the form of sodium and potassium chlorophenate salts, usually as aqueous solutions of approximately 0.15 percent pentachlorophenate. Most chlorophenate products are mixtures of chlorophenols with one compound normally present in greater amounts than others. For example, one such product has approximately 14 percent pentachlorophenol, 8 percent tetrachlorophenol, 6 percent other chlorophenols and the remainder composed of inert ingredients. Such a solution would then be diluted with water for surficial wood treatment purposes.

At sawmills where surficial treatment is used to prevent fungal damage, rough sawn lumber may be treated by either dipping it into large tanks containing the preservative solution, or by spraying the solution on the wood after sawing. The wood is then set aside and allowed to dry. Provisions may or may not be made to recover the excess treatment solution, and in many cases it is lost to the soil in the sorting or drying areas. A recent improvement employed by some operations is the construction of treating and drying facilities with sloping floors and sumps, which allow the excess treatment solution to be collected and either recycled or disposed of.

Other wood preservation methods may use pressure treatment methods, usually a pressurized retort, to more fully saturate the cells of the wood for more complete and longer lasting protection; utility poles are commonly treated in this manner. Several carriers have been used in pressure retort operations; for example, PCP may be dissolved in oil, liquified petroleum gas

and isopropyl ether mixture, mineral spirits or methylene chloride (Morgan, 1986). In some cases the PCP may be dissolved in a solvent such as diesel oil, and wood products may be soaked in the solution without the use of a pressure system.

Over time there is an accumulation of treating process residuals in the form of sediments and sludge in most systems. In sawmill dip tank operations, these residuals consist of sawdust and other debris which collect at the bottom of the treatment tank and must be periodically removed. Disposal of these wastes is currently a problem, since they contain CDDs and CDFs at much higher levels than found in the treatment solution. Since CDD and CDF containing wastes are no longer accepted at California landfills due to potential liability problems (see Chapter 7; Criteria, Standards, and Regulations), this waste must be either temporarily stored on-site or disposed of outside of California. (The U.S. Hazardous and Solid Waste Amendments of 1984 specifically banned landfilling of "dioxin-containing wastes" effective November 8, 1986. However, the Amendments allowed issuance of a national variance on the ban for up to 2 years. Citing the lack of disposal and treatment options, the U.S. EPA issued a variance that will expire on November 8, 1988.)

One available means of disposal has been to burn the sludge on-site by various low-temperature methods, such as in a tepee burner. Burning under such conditions not only does not reliably destroy CDDs and CDFs, but also produces them from precursor chlorophenol compounds. In addition, it releases them to the environment adsorbed to the soot (Tiernan et al., 1983). Burial of these wastes on-site has also been a common practice. Recently, as a temporary measure, on-site storage and containment of these materials in drums has been recommended as an interim measure, but a long-term solution is still needed.

CASE STUDIES OF CONTAMINATED SITES IN CALIFORNIA

Annual production of PCP in the United States is estimated by U.S. EPA (Esposito et al., 1980) to be about 53 million pounds annually. In California, over 2 million pounds of PCP were sold in 1983 (CARB and CDHS, 1986). Approximately 90 percent of this amount was used in wood treatment facilities employing pressure treatment methods (pentachlorophenol), and 10 percent was used in sawmill operations (pentachlorophenate salts).

Three examples of contamination occurring as a result of wood treatment operations are described, with each in a different stage of the evaluation and cleanup process. They are also fairly representative of several additional sites in the State which are awaiting further investigation. California currently has approximately 10 wood treatment facilities and 86 sawmills in operation (CARB and CDHS, 1986), along with a number of facilities which are no longer functional. A recent consultant's

report to the CARB contains an inventory of California sawmills and wood treatment plants using chlorinated phenols as of December 1986 (Chinkin et al., 1987). According to the report, five pressure treatment plants account for 98 percent of current chlorophenol use in wood treatment. The remaining two percent reflects tetrachlorophenate use at four sawmills. This represents a decline in chlorophenol use at sawmills since 1983.

Oroville Wood Treatment Site

A wood treatment facility near Oroville, California is currently being evaluated for soil and ground water contamination resulting from long-term wood preservative use. This 200 acre site has been associated with the lumber industry since about 1920. Both PCP and creosote have been found in soil and ground water both on and off-site (U.S. EPA, 1986a).

The investigation, still in its preliminary stages, has determined levels of PCP in the soil of at least 10 ppm with creosote also present. The depth to water is about 30 feet, and levels of PCP in the ground water below the site range up to 15,000 ppb. Ground water flows in a south-southwest direction, and private wells adjacent to and downgradient from the site have levels of PCP ranging up to 4000 ppb. To the south of the site a plume of contamination extending at least two miles and containing levels of PCP up to 2000 ppb has been detected. The depth to water in this second area is 90 to 120 feet (U.S. EPA, 1986a).

While approximately 30 domestic wells in this rural area have been found to be contaminated with PCP, no CDDs or CDFs have been detected in ground water. Residents have complained of adverse health effects they believe are related to the contamination, such as diarrhea and skin disorders, and an alternate domestic water supply has been provided. Recovery wells have been constructed in the area to the southwest of the site in an effort to reduce contaminant levels (U.S. EPA, 1986a).

In compliance with a U.S. EPA work plan, surface water, ground water, sediment and soil core studies will be performed along with other hydrogeological testing. The first phase of soil and ground water study is expected to better define the extent of contamination (U.S. EPA, 1986a).

Selma Wood Treatment Site

A facility near Selma in Fresno County has also been associated with soil and water contamination resulting from wood preserving operations. This 18 acre facility has been in operation since about 1936, and is bordered by residential, agricultural and industrial areas, some located as close as one-fourth of a mile. (U.S. EPA, 1986d).

A variety of wood preserving chemicals have been used at this site during its history, with those used since 1965 including chromated copper arsenate (CCA), copper-8-quinolinolate, and pentachlorophenol dissolved in ketone solvents, diesel fuel or mineral spirits. During 1982 it was estimated that, using pressure treating methods, about 1,000 gallons of 5 percent PCP solutions and 3,000 to 4,000 gallons of a 1.5 percent CCA solution were used daily to treat lumber products, including utility poles, grape stakes, and fence posts (CVRWQCB, 1982).

During its operation the disposal of treatment related wastes was accomplished by discharge into dry wells, into an unlined pond, runoff into drainage ditches, to the open ground and into a sludge pit. Relatively recent improvements include disposal of drummed waste, such as sludge off-site, and containment of contaminated surface runoff from the treatment area (CVRWQCB, 1982).

The Central Valley Regional Water Quality Control Board first sampled the site in 1971, with the Department of Health Services becoming involved in 1983, and the EPA assuming enforcement responsibility in 1984. The results of this sampling are summarized in Table 6.1.

The aquifers and aquitards in the area are composed of continuous and discontinuous layers of unconsolidated gravel, sand, silt and clay, with the depth to water approximately 30 feet. To the west of the site is found the Corcoran Clay layer, which divides the ground water into a confined and unconfined aquifer system. Because the facility is located on the eastern side of the Central Valley in what may be a recharge zone for those aquifers to the west (ground water flows to the southwest from the site), there is concern about off-site migration since the vertical and horizontal extent of soil and ground water contamination has not been completely defined (U.S. EPA, 1986d). Currently, the U.S. EPA is conducting a sampling program as part of its investigation to better define the vertical and lateral extent of contamination both on and off site.

The CDD and CDF results for 2 of the 25 soil samples and for both pressurized retort effluent samples taken in April 1986 are shown in Table 6.2. The CDD and CDF levels in the soil samples are similar to those of the retort effluent samples; however, some tetra- and pentaCDFs and pentaCDDs were detected in soil samples which were not present in the retort effluent samples.

Visalia Wood Treatment Site

Ground water contamination resulting from the use of PCP and creosote at a facility where electrical power poles were treated has been followed and documented since 1973. This site is located at Visalia, California, where a dip tank containing PCP

TABLE 6.1

SELMA PRESSURE TREATMENT PLANT CONTAMINATION SUMMARY
(adapted from U. S. EPA 1986c)

	PCP (ppm)[1/]
Drinking Water Standard	1.0[2/]
Surface Water Sampling Results Range	0.24-2.3
Soil Sampling Range	
Surface to 2 ft. depth	0.06-4,500
2 ft. to 5 ft.	<0.8 -3,100
5 ft. to 10 ft.	0.1 - 600
10 ft. to 20 ft.	2.6 - 41
Greater than 20 ft.	<0.5 - 1.2
Ground Water Sampling Results Range	0.002

1/ Parts per million
2/ State of California Action Level

TABLE 6.2

CDD AND CDF CONCENTRATIONS (WET WEIGHT)
IN SOIL AND RETORT EFFLUENT AT SELMA
PRESSURE TREATMENT SITE
(Compiled from U. S. EPA, 1986c)

	Soil Samples		Retort Effluent Samples	
	A	B	A	B
CDD (ppb):				
tetra	ND[a/]	ND	ND	ND
penta	5.8*	ND	ND	ND
hexa	324*	383*	380*	275*
hepta	3,970*	5,100*	18,500*	19,400*
octa	13,300	14,900	10,500	110,000
CDF (ppb):				
tetra	2.3*	3.1*	ND	ND
penta	72.5*	80*	ND*	ND
hexa	601*	711*	917*	767*
hepta	1,410*	1,660*	11,300*	10,500*
octa	2,990	6,200*	43,700	49,200

[a/] Not detected
* Approximate values

dissolved in number 2 diesel oil was discovered leaking in late 1972. The tank was replaced and an investigation initiated to determine the extent of soil and ground water contamination. The plant used PCP for pole treatment from 1968 to 1980, when operations came to an end.

In this area ground water is contained in two saturated zones separated by an aquitard, a feature characteristic of the San Joaquin ground water basin (Figure 6.1; DWR, 1982). At the site, depth to water in the shallow unconfined aquifer is approximately 30 feet, with the aquitard varying between 10 and 20 feet in thickness encountered at about 65 feet confining the deeper aquifer (SCE, 1985d).

The unsaturated and unconfined layers, considered moderately permeable, are composed of alluvial silts and fine sands near the surface, progressing through medium and coarse grained sands to pebble gravel at the upper boundary of the aquitard (SCE, 1983). The aquitard is composed of silts and clays of low hydraulic conductivity and is considered a leaky, saturated confining layer for the deep aquifer, which consists of coarser grained more permeable alluvial deposits. Ground water in the shallow aquifer flows in a generally south-southwest direction, following a gradient of about 17 feet per mile. Flow in the deep aquifer is in a generally west-southwest direction, following a gradient of about 15 feet per mile (SCE, 1984). The deep aquifer is confined at its base by relatively impermeable beds, and is widely used as a source of drinking water by many in the area, including the City of Visalia.

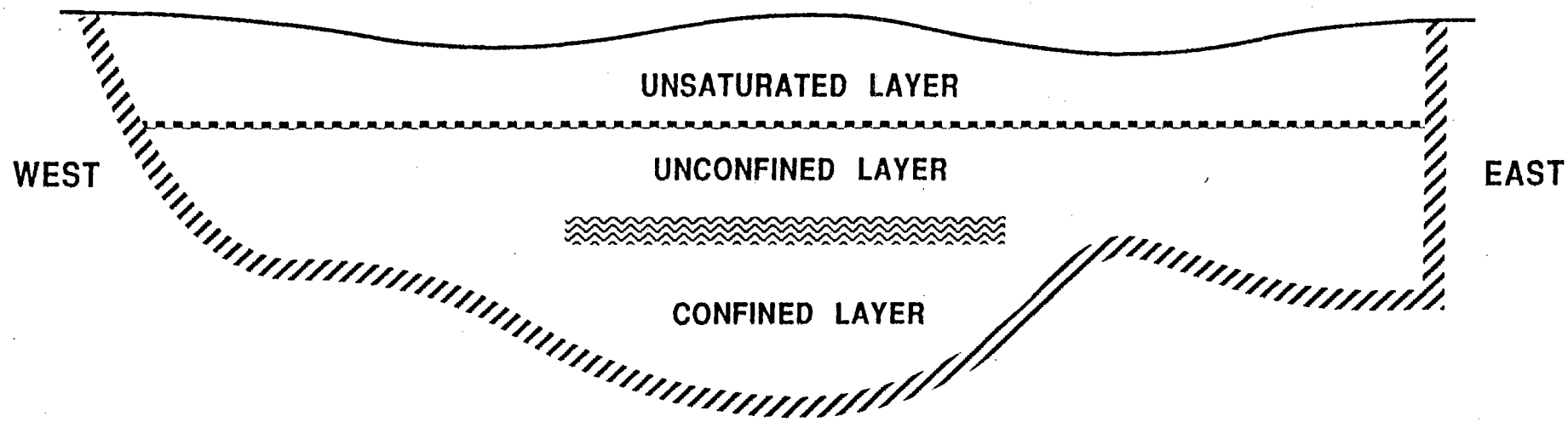
Since the leak was discovered, a series of monitoring and recovery wells have been installed, as both the shallow and deep aquifers have been contaminated with PCP, creosote, CDDs and CDFs. To inhibit downgradient movement of the contaminants off-site, a bentonite-cement slurry wall has been built below the surface. This barrier surrounds the shallow aquifer beneath the site, and extends from the surface to its lower boundary.

Contaminant levels in ground water have fluctuated significantly since the investigation began in 1973, with the highest levels reached in 1977, (Table 6.3). During this same period PCP was detected in monitoring wells 600 feet to the south of the site at levels of 0.3 to 37 ppm, and also 1600 feet to the soutwest at levels of 0.007 to 2 ppm, with creosote also present in both cases (SCE, 1983).

To reduce contaminant levels and prevent further migration away from the site, ground water has been pumped from the shallow aquifer since 1975, and from the deep aquifer since 1976. Over time, additional monitoring and recovery wells have been added. The water has been discharged to the City of Visalia Water Conservation Plant. CDDs and CDFs were found in the ground water

FIGURE 6.1

TYPICAL CROSS SECTION OF THE
SAN JOAQUIN VALLEY GROUNDWATER BASIN
(ADAPTED FROM DWR, 1982)



LEGEND

Aquitard (Confining Clay Layer)

Water Table

No Flow Boundary

TABLE 6.3

CONCENTRATIONS OF PCP, CDDs AND CDFs IN SOIL AND WATER AT VISALIA POLE TREATMENT SITE*

Contaminant	Shallow Unconfined Aquifer 1977	1984	1985	Deep Confined Aquifer 1977	1984	1985	Soil A 1985		Soil B 1985		Sewage Sludge 1985
							Lab 1	Lab 2	Lab 1	Lab 2	
PCP (ppm)	44,000	17.0	16.7	6.3	4.5	1.5	-		-		-
Creosote (ppm)	73,000	6.2	6.5	270	47.0	21.2	-		-		-
CDD (ppb):											
Tetra	-[a/]	<0.001	<0.00054	-	<0.001	<0.0007	<0.2	<0.07	<0.1	<0.05	<0.03
Penta	-	<0.001	<0.0013	-	<0.001	<0.00023	<0.2	<0.23	0.1	<0.17	<0.17
Hexa	-	<0.001	<0.00064	-	0.0025	0.0027	21	12	125	240	6.5
Hepta	-	<0.001	<0.0011	-	0.113	0.210	260	490	1730	500	110
Octa	-	0.049	0.0069	-	1.140	0.870	1810	2300	2388	1700	86
CDFs (ppb):											
Tetra	-	<0.001	<0.00044	-	<0.001	<0.00034	<0.2	<0.05	<0.1	<0.05	<0.04
Penta	-	<0.005	<0.00063	-	<0.01	<0.00051	0.4	2.3	3.8	9.3	<0.07
Hexa	-	<0.001	<0.0013	-	0.030	0.014	48	89	366	180	3.7
Hepta	-	<0.001	<0.0023	-	0.15	0.210	141	670	1047	600	12.0
Octa	-	<0.001	<0.0044	-	0.15	0.220	76	540	331	190	6.7

a/ Not Analyzed

* Compiled from SCE 1983, 1984, 1985a, 1985b, 1985c, 1985d

of both shallow and deep aquifers in 1984, and as shown in Table 6.3, the levels increased in 1985. Further investigation of soils from the site both at the surface and to a depth of eight inches detected significant levels of CDDs and CDFs, mostly hexa-, hepta-, and octa- isomer groups, with pentaCDFs also present.

Soil cores from the construction of additional monitoring wells in 1984 have provided data on the vertical distribution of PCP, creosote, CDDs and CDFs in the shallow aquifer (Table 6.4). The hexa-, hepta-, and octa- isomer groups again predominate. While no tetraCDDs or pentaCDDs were detected at any depth, tetraCDFs and pentaCDFs were present. The soil corings were taken from the location of the leaking tank, and began at a depth of 30.5 feet, as the contaminated soil above previously had been removed and replaced with clean fill. The aquitard material separating the two aquifers was also sampled at several locations at the site to determine if contaminants were able to penetrate this barrier. PCP, creosote, and low levels of hexa- through octaCDDs and penta- through octaCDFs (Table 6.4) were found within the aquitard under a location where treated poles had been stored.

Water recovered from both aquifers containing PCP, creosote, CDDs and CDFs has been discharged to the Visalia Water Conservation Plant since pumping began. PCP and creosote have been detected in the plant influent, effluent and sludge. CDDs and CDFs have also been detected in the influent, with the highest levels found in the sludge shown on Table 6.3. CDDs and CDFs have not been detected in the plant effluent.

Sludge from this plant is used as a soil amendment by farms and residents in the area, and a recent study (SCE, 1986) determined that sludge stockpiles at the distribution point contained CDDs and CDFs at levels similar to sludge from the water conservation plant. In this study sludge application rates ranged from 2.3 tons per acre to 259 tons per acre, and levels of CDDs and CDFs in the soils in these areas appeared to correlate with the application rate. While no tetraCDDs were found in the soil samples, tetraCDFs were present. Approximately 20 percent of the total tetraCDFs present were estimated to be the 2,3,7,8-tetraCDF isomer.

In 1985 a pretreatment system was installed at the site to remove contaminants from the extracted ground water before being received by the water conservation plant. The water is first passed through filters designed to trap CDD and CDF containing particulates, and then through carbon beds to remove PCP along with other organics. The system is designed to allow ground water to eventually be pumped directly into a nearby creek after treatment, bypassing the water conservation plant. Only trace levels of PCP (0.15 ppb) have been found after such treatment, with creosote, CDDs and CDFs not detected. A request based on this system's performance is before the Regional Water Quality Control Board to allow such a discharge (SCE, 1985c, 1986).

TABLE 6.4

VERTICAL DISTRIBUTION OF SOIL AND AQUIFER CONTAMINANTS AT VISALIA SITE[a]

Depth (ft)	PCP (ppm)	Creosote (ppm)	CDD (ppb)					CDF (ppb)				
			Tetra	Penta	Hexa	Hepta	Octa	Tetra	Penta	Hexa	Hepta	Octa
Soil Core Samples												
30.5	61	3700	<0.01	<0.08	1.2	68	460	1.3	<0.38	36	100	180
35.5	48	1500	<0.02	<0.05	0.3	30	320	<0.02	<0.04	13	36	60
40.5	120	ND[b]	<0.02	<0.06	7.0	340	1700	<0.02	0.33	49	455	200
40.5	5.9	ND	<0.02	<0.05	1.6	41	370	<0.01	<0.02	4.2	32	26
46.5	-[c]	-	<0.02	<0.06	4.7	100	720	<0.01	0.19	100	100	41
54.5	14	160	<0.12	<0.3	<0.33	2.8	63	<0.18	<0.27	<0.42	2.8	<2.2
Aquitard Profile Samples												
45.0	0.16	620	-	-	-	-	-	-	-	-	-	-
50.0	0.27	250	-	-	-	-	-	-	-	-	-	-
52.0	1.3	900	-	-	-	-	-	-	-	-	-	-
54.0	0.82	110	-	-	-	-	-	-	-	-	-	-
56.0	3.6	1100	-	-	-	-	-	-	-	-	-	-
58.0	8.0	2000	<0.0092	<0.036	2.4	216	327	<0.010	0.27	9.5	197	167

[a] Data from SCE, 1984, 1985a
[b] Not Detected
[c] Not Analyzed

Progress in removing contaminants from both aquifers has generally been good; in most cases a greater than 90 percent reduction of peak levels has been seen. Since about 1980, levels have been erratic from one analysis to the next, and the level of improvement somewhat uncertain, particularly for wells on the site. Contaminant levels in wells located further away from the site in the path of the plume have been more consistent, and do indicate a downward trend. The proposed level of clean up for ground water at the extraction wells before treatment is 1 ppm total phenols, 30 ppb for PCP, and below detection limits for creosote, CDDs and CDFs.

CHAPTER 7: CALIFORNIA WATER RESOURCES CONTROL BOARD STUDY

The State Board study described in this chapter proceeded in three stages over a two year period: (1) analysis for presence of pentachlorophenol (PCP); (2) preliminary screening for CDDs and CDFs; and (3) analyses for specific CDD and CDF compounds of toxicological concern. The study began by examining potential contamination by PCP and related chlorophenols from wood treatment operations. While sampling for PCP at these facilities, the State Board learned of a Swedish study (Levin et al., 1976) that reported high levels of CDFs detected in sludges from sawmill dip tanks.

When significant levels of PCP contamination were found at sampling sites, the State Board initiated a preliminary screening for CDD and CDF isomer groups to determine if the Swedish CDF findings were representative of California conditions. The screening confirmed the presence of both CDDs and CDFs. These findings were presented to a California interagency task force which recommended that (1) future samples be split between different laboratories for verification and (2) if possible, analyses should be performed for 2,3,7,8-chlorinated compounds since these are the specific congeners of toxicological concern.

Based on these recommendations, the State Board staff designed a two phase study of congener specific analyses. These results are presented in detail later in the chapter and form the basis for the hazard evaluation strategy described in Chapter 8.

ANALYTICAL METHODS

The search for CDDs and CDFs in various industrial, occupational and environmental settings has pushed analytical methodology to its limits, and the gas chromatography-mass spectrometry (GC-MS) methods in current use represent the state-of-the-art. The extreme toxicity of 2,3,7,8-tetraCDD at very low levels of exposure has determined the need for very sensitive and specific methods of analysis, and has lowered the limit of detection from 1 ppm in 1969 to sub-parts per trillion levels today (NRCC, 1984). Analytical methods are discussed in detail by the U.S. EPA (Esposito et al., 1980; U.S. EPA, 1985b, 1986a), NRCC (1981), and Tiernan (1983); an overview is presented in Appendix E, with the analytical methods used by State Board contract labs described in Appendices F and G.

STATE BOARD ISOMER GROUP STUDY

This preliminary study was designed to determine if CDDs and CDFs were present as a result of using chlorinated phenols for wood treatment. Included in the study were sawmills or wood treatment

facilities both currently operational and some that had discontinued operations. Operating sawmills were located in Shasta County, Tehama County, and Trinity County. Abandoned sawmills in Glenn County, Humboldt County, and a combined sawmill-wood treatment plant in Sonoma County which had discontinued operations were also part of the study. Samples were obtained of the chlorophenol products in use, the dilute dip tank solution, accumulated sludge from the bottoms of these tanks, and of the soil in the area of treatment operations.

Chlorophenol Products

Samples of two pentachlorophenates and one tetrachlorophenate products used in sawmill operations were obtained and analyzed for chlorophenol, CDDs and CDFs. The results shown in Table 7.1 demonstrate the high variability between different lots, which is common to products from different manufacturers and processes. The higher chlorinated Cl_6 to Cl_8 CDDs and CDFs are present in the largest amounts, which is typical (see Appendix D, Table D.1).

Of these higher chlorinated CDDs and CDFs, the hexa isomer group is of chief toxicological concern, with the isomers chlorinated in the 2,3,7, and 8 positions having about 1/100 of the acute toxicity of 2,3,7,8-tetraCDD in the guinea pig. The mixture of 1,2,3,6,7,8- and 1,2,3,7,8,9-hexaCDDs has also been determined to be an animal carcinogen and potential human carcinogen.

The results, shown in Table 7.1, are similar to those obtained in other studies, especially with regard to the relative abundance of isomer groups in products from different manufacturers. When pentachlorophenol and pentachlorophenate (PCP salts) products are compared, the chlorophenates generally contain greater amounts of CDFs than the pentachlorophenols, which usually have greater amounts of CDDs compared with pentachlorophenates.

Product and Soil Residues

Results of the isomer group analyses are presented in Table 7.2. The samples of dip tank solution, sludge and soil all show the same general pattern of CDD and CDF isomer group distribution seen in the products, with the higher chlorinated Cl_6 to Cl_8 groups present at the highest levels. CDDs and CDFs appear to be concentrated in the sludge from the dip tanks. The levels in the dried sludge are anywhere from about 10 to almost 1,000 times greater than those of the dip tank liquid, with hepta- and octa-CDFs showing the greatest enrichment.

TABLE 7.1

CDD AND CDF CONCENTRATIONS IN COMMERCIAL CHLOROPHENOL PRODUCTS[a/]

	Tetrachlorophenate (Sodium)	Pentachlorophenate "A" (Sodium)	Pentachlorophenate "B" (Sodium)
TetraCP (ppm)	140,000	140,000	77,000
PCP (ppm)	31,000	170,000	150,000
CDDs (ppb):			
Tetra	<1.0	<0.5	16
Penta	238	11	1,400
Hexa	1,100	4,800	14,000
Hepta	614	88,000	64,000
Octa	700	216,000	69,000
CDFs (ppb):			
Tetra	1,060	190	2,800
Penta	22,100	380	3,400
Hexa	17,600	1,900	18,000
Hepta	3,000	4,100	18,000
Octa	62	2,900	840

[a/] State Water Resources Control Board data developed for the present report.

TABLE 7.2

CDD AND CDF CONCENTRATIONS IN SOIL AND PRODUCT RESIDUES RELATED TO CHLOROPHENOL USE[a/]

	Shasta County Sawmill Dip Tank Liquid	Shasta County Sawmill Dip Tank Sludge	Glenn County Sawmill Dip Tank Wet Sludge	Glenn County Sawmill Dip Tank Dry Sludge	Tehama County Sawmill Dip Tank Liquid	Trinity County Sawmill Dip Tank Sludge
TetraCP(ppm)	1,700	4,000	300	37,000	11,000	2,300
PCP (ppm)	2,200	5,700	880	160,000	3,700	2,600
CDDs (ppb)						
Tetra	<0.002	1.7	0.57	51	<0.34	<0.35
Penta	0.2	16	19	2,000	6.4	84
Hexa	7.7	799	360	13,000	86	2,300
Hepta	112	3,066	1,200	23,000	111	13,000
Octa	352	3,066	3,500	7,400	428	26,000
CDFs (ppb)						
Tetra	0.84	54	21	5,600	32	110
Penta	3.7	259	92	3,600	106	2,200
Hexa	4.3	1,143	140	12,000	936	1,600
Hepta	0.37	369	350	5,700	90	1,500
Octa	1.3	1,066	17	250	90	65

[a/] State Water Resources Control Board data developed for the present report.

TABLE 7.2 (continued)

CDD AND CDF CONCENTRATIONS IN SOIL AND PRODUCT RESIDUES RELATED TO CHLOROPHENOL USE[a/]

	Tehama County Sawmill Soil	Trinity County Sawmill Soil	Humboldt County Sawmill Soil	Sonoma County Wood Treatment Plant Soil
TetraCP (ppm)	3,700	1,400	-	-
PCP (ppm)	6,400	1,600	-	260
CDDs (ppb):				
Tetra	<2.2	<0.48	<0.088	<0.014
Penta	11	5.5	<0.20	<0.18
Hexa	180	245	9.4	44
Hepta	185	3,100	37	4,400
Octa	977	1,600	188	1,500
CDFs (ppb):				
Tetra	48	100	12	0.23
Penta	105	45	33	9.6
Hexa	1,593	540	126	230
Hepta	229	730	183	2,100
Octa	242	120	41	1,200

[a/] State Water Resources Control Board data developed for the present report.

CONGENER SPECIFIC SURVEY

Background

The CDD and CDF isomer group data obtained from sampling sawmill soils and sludge tanks indicated that these contaminants would be of toxicological concern if a significant fraction were chlorinated at the 2,3,7, and 8 molecular positions. Several approaches can be used that either estimate or directly measure the concentrations of 2,3,7,8 congeners present. From a health standpoint, the most conservative estimate is to assume that all tetra through hepta CDDs and CDFs are chlorinated at positions 2,3,7, and 8. A second method, which has been used to estimate toxicity in municipal solid waste emissions (CARB and CDHS, 1986), assumes equal distribution for each isomer within an isomer group. For example, the level of 2,3,7,8-tetraCDD, as one of 22 possible isomers in the tetraCDD isomer group, would be assigned 1/22 or 4.5 percent, of the total tetraCDD reported in a sample. A third method is direct measurement of individual CDD and CDF congeners present in a sample. The latter is difficult, time-consuming, expensive, and truly state-of-the-art chemistry, particularly in complicated media such as soil and sludge.

Members of the State Board and its management were informed of the sawmill sampling data for CDDs and CDFs in August 1984. The Secretary of Environmental Affairs and representatives of several state agencies were briefed during the following October. The consensus was that, while the CDD and CDF findings were provocative, the results should be considered tentative until confirmed by another laboratory. Further, it was recommended that the State Board attempt to determine if the congeners of greatest concern (those CDDs and CDFs chlorinated at the 2,3,7, and 8 positions) were present in sawmill residues where tetrachlorophenol and pentachlorophenol had been used. Table 7.3 lists the seven CDDs and ten CDFs that contain the 2,3,7,8 pattern of chlorination. The State Board's congener-specific sampling program focused on these 17 CDDs and CDFs and did not analyze for non-2,3,7,8 congeners.

State Board staff planned a two phase program of analysis. Phase 1 would examine a limited number of samples from two sawmills for congener-specific analysis, utilizing samples previously collected and identified as containing high isomer group levels of CDDs and CDFs. If Phase 1 identified the presence of 2,3,7,8 congeners of CDDs and CDFs, then a second more extensive phase would examine samples from two additional sites, a third sawmill and a wood pressure treatment plant. The locations of sites sampled in Phases 1 and 2 and the number of samples taken at each site are shown in Table 7.4.

TABLE 7.3

2,3,7,8-CHLORINE SUBSTITUTED CONGENERS OF CDDs AND CDFs

Isomer Group	Total Isomers in Isomer Group	Number of Isomers in Isomer Group with 2,3,7,8 Substitution	Specific Isomers
CDDs:			
Tetra-	22	1	2,3,7,8-tetraCDD
Penta-	14	1	1,2,3,7,8-pentaCDD
Hexa-	10	3	1,2,3,4,7,8-hexaCDD
			1,2,3,6,7,8-hexaCDD
			1,2,3,7,8,9-hexaCDD
Hepta-	2	1	1,2,3,4,6,7,8-heptaCDD
Octa-	1	1	1,2,3,4,6,7,8,9-octaCDD
Total tetra through octaCDD congeners	49	7	
CDFs:			
Tetra-	38	1	2,3,7,8-tetraCDF
Penta-	28	2	1,2,3,7,8-pentaCDF
			2,3,4,7,8-pentaCDF
Hexa-	16	4	1,2,3,4,7,8-hexaCDF
			1,2,3,6,7,8-hexaCDF
			1,2,3,7,8,9-hexaCDF
			2,3,4,6,7,8-hexaCDF
Hepta-	4	2	1,2,3,4,6,7,8-heptaCDF
			1,2,3,4,7,8,9-heptaCDF
Octa-	1	1	1,2,3,4,6,7,8,9-octaCDF
Total tetra through octaCDF congeners	87	10	

TABLE 7.4

STATE WATER RESOURCES CONTROL BOARD PROGRAM FOR CONGENER SPECIFIC ANALYSIS OF CDDs AND CDFs

A. Phase I

1. Sawmill A (Trinity County): 2 samples

   a. Commercial sodium pentachlorophenate
   b. Dip tank sludge

2. Sawmill B (Glenn County): 2 samples

   a. Wet dip tank sludge
   b. Dry mix tank sludge

B. Phase II

1. Sawmill C (Humboldt County): 4 samples

   a. Commercial potassium tetrachlorophenate
   b. Dip tank liquid
   c. Dip tank sludge (2 samples)

2. Wood Treatment Plant (San Joaquin County): 4 samples

   a. "Bloom"
   b. "Commercial"--recycled treatment material
   c. Soil at retort
   d. Sump liquid

Phase 1 Sampling Results

The samples for Phase 1 were collected at two non-functioning sawmills. Sawmill A, which has been dismantled, was located on the Trinity River in Trinity County. The lumber dip tank was located in a concrete-block structure covered by a sheet metal roof. The dip tank itself had been constructed by walling off a portion of one end of the building with additional cinder blocks so that two of the tank walls were actually part of the building's exterior walls. Four samples consisting of the commercial PCP formulation, dip tank liquid, dip tank sludge, and soil were collected and analyzed for CDD and CDF isomer groups. These results indicated that the commercial PCP and dip tank sludge were highest in CDDs and CDFs. These two samples, as well as two sludge samples from Sawmill B, were subsequently split two ways and sent to participating laboratories in California and Sweden. Since the samples were not split homogeneously, differences in laboratory results beyond expected analytical variation are possible.

The results for Sawmill A are shown in Appendix H, Results of State Board 2,3,7,8 Congener Specific Analyses, Table H.1. The 2,3,7,8-chlorinated CDD congeners present include 1,2,3,7,8-pentaCDD; 1,2,3,6,7,8-hexaCDD; and 1,2,3,4,6,7,8-heptaCDD. Chlorinated dibenzofuran congeners include 2,3,7,8-tetraCDF; 1,2,3,7,8-pentaCDF; 2,3,4,7,8-pentaCDF; 1,2,3,6,7,8-hexaCDF; 1,2,3,7,8,9-hexaCDF; and 1,2,3,4,6,7,8-heptaCDF. Table 7.5 is a summary of results that shows the percentage of 2,3,7,8-chlorinated isomers present within a given isomer group.

Sawmill B was located in Glenn County. Based on CDD and CDF isomer group results, two samples were selected for congener-specific analysis: a mixture of liquid and sludge from the dip tank and a dry sludge from an elevated wood preservative mix tank. The mix tank sludge contained approximately 10 ppb 2,3,7,8-tetraCDD (Table H.2 of Appendix H). However, without a record of chemicals used in the mix tank, the source of 2,3,7,8-tetraCDD can not be determined. The mix tank also contained approximately 200 ppb of 1,2,3,7,8-pentaCDD and 4,000 ppb 1,2,3,6,7,8-hexaCDD.

Phase 2 Sampling Results

Samples for Phase 2 were obtained at two sites: Sawmill C in Humboldt County and a wood treatment plant in San Joaquin County. Four samples were taken at each site and split for analysis by laboratories in California and Illinois. Sawmill C is a functioning lumber mill that had been using a unit dip tank for 3-1/2 years. This below ground level tank is housed in a special facility designed to contain wood preservatives totally within the building and represents current thinking on best management

TABLE 7.5

CDD AND CDF CONGENER SPECIFIC ANALYSIS, PHASE 1: SAWMILLS A AND B

(Summary of isomer group data and percent of isomer group consisting of 2,3,7,8 chlorinated isomers; average of two laboratories except that, where differences exceed 5X, both values are reported).

	Commercial Na-PCP Formulation	Sawmill A Sludge	Sawmill B Liquid Sludge	Sawmill B Dry Sludge
CDDs (ppb):				
TetraCDD	-	-	-	8.4 60
2,3,7,8	-	-	-	8.3 11
% 2,3,7,8 of total tetraCDD	-	-	-	99% 18%
PentaCDD	222	68	25 246	1,009
1,2,3,7,8	26	<15.9 10	34 14	199
% 2,3,7,8 of total pentaCDD	12%	- 9%	136% 5.7%	20%
HexaCDD	9,850	3,115	500	7,400
total 2,3,7,8 (3 isomers)	3,825	1,795	258	4,187
% 2,3,7,8 of total hexaCDD	39%	58%	50%	57%
HeptaCDD	70,000	39,200	3,200	18,000
1,2,3,4,6,7,8	30,900	23,500	2,050	11,015
% 2,3,7,8 of total heptaCDD	44%	60%	64%	61%
CDFs (ppb):				
TetraCDF	1,436	383	78	1,997
2,3,7,8	201	105	17	95
% 2,3,7,8 of total tetraCDF	14%	27%	22%	4.8%
PentaCDF	8,200	4,035	575	11,050
2,3,7,8 (2 isomers)	614	220	50	280
% 2,3,7,8 of total pentaCDF	7.5%	5.5%	8.7%	2.5%
HexaCDF	49,000 7,200	8,600	1,530 900	7,900
2,3,7,8 (4 isomers)	705 500	373	45 ND	292
% 2,3,7,8 of total hexaCDF	1.4% 6.9%	4.3%	2.9% -0-	3.7%
HeptaCDF	91,000 9,700	8,650	830	3,550
1,2,3,4,6,7,8	6,344 3,900	2,635	373	1,510
% 2,3,7,8 of total heptaCDF	7% 40%	30%	45%	43%

of dip method wood treatment. For the first two years of unit dip tank operation, Sawmill C had used a formulation containing 15 percent each of pentachlorophenate and tetrachlorophenate.

That formulation then was replaced by a preservative containing approximately 22 percent potassium tetrachlorophenate and 6 percent potassium pentachlorophenate, a mixture used for 1-1/2 years at the time of sampling. One by-product in the unit dip tank has been the accumulation of sludge that must eventually be removed. Samples were taken of the current commercial formulation (22 percent K-tetraCP and 6 percent K-PCP), the dip tank liquid, and two sludge samples, one from the tank center and the second from a corner. Results from the two laboratories are summarized in Table H.3 of Appendix H. One laboratory reported approximately 7.0 ppb of 2,3,7,8-tetraCDD in a sludge sample, but the finding was not confirmed by the second laboratory (Table 7.6 presents the condensed results of Table H.3). No 1,2,3,7,8-pentaCDD was found. The predominant hexaCDD was the 1,2,3,6,7,8-isomer. As shown in Table 7.6, the predominant 2,3,7,8 CDFs of the toxicologically significant tetra-, penta-, and hexa- isomer groups were 2,3,7,8-tetraCDF and the two 2,3,7,8-pentaCDF isomers.

The wood treatment plant uses a pressurized retort system to treat poles and other wood with pentachlorophenol dissolved in butane. The label on the commercial PCP indicated that it contained 86 percent PCP and 10 percent other chlorinated phenols. When treated wood is removed from the retort under atmospheric pressure, solution oozes out and collects as crystals on the wood surface. The crystals are referred to as the "bloom". A sample of the bloom was scraped off for analysis.

The other three samples consisted of: (1) "commercial", a recycled wood preservative material that is combined with butane and reused in the treatment process; (2) soil from the mouth of the pressure retort system; and (3) liquid from a nearby sump that, when sampled, was characterized as "probably a mixture of penta and oil". Results are summarized in Table H.4 of Appendix H.

Only the bloom contained detectable levels of 1,2,3,7,8 pentaCDD. Table 7.7 provides information on the percent of 2,3,7,8 congeners present within CDD and CDF isomer groups.

SUMMARY AND DISCUSSION

The analytical methodology developed for 2,3,7,8-tetraCDD has progressed from the ability to analyze for isomer groups of CDDs and CDFs to the detection and quantification of individual congeners requiring the very latest developments in technology. Gas chromatography-mass spectrometry is the method of choice for CDD and CDF analysis, and was used by State Board contract

TABLE 7.6

CDD AND CDF CONGENER SPECIFIC ANALYSIS, PHASE 2: SAWMILL C

(Summary of isomer group data and percent of isomer group consisting of 2,3,7,8 chlorinated isomers, average of two laboratories except that, where differences exceed 5X, both values are reported.)

	Commercial Tetrachloro-Phenate Formulation	Sawmill Dip Tank Liquid	Sawmill Dip Tank Sludge[1]	Sawmill Dip Tank Sludge[1]
CDDs (ppb):				
TetraCDD	ND	0.5	ND	7.4[2]
2,3,7,8	ND	ND	ND	ND
% 2,3,7,8 of total tetraCDD	-	0	-	0
PentaCDD	256 21	1.0	30	36
1,2,3,7,8	ND ND	ND	ND	ND
% 2,3,7,8 of total pentaCDD	0 0	0	0	0
HexaCDD	1,240	12	404	477
total 2,3,7,8 (3 isomers)	509	6.0	206	244
% 2,3,7,8 of total hexaCDD	41%	50%	51%	47%
HeptaCDD	1,083	20	1,472	1,582
1,2,3,4,6,7,8	688	12	886	937
% 2,3,7,8 of total HeptaCDD	64%	60%	60%	49%
CDFs (ppb):				
TetraCDF	1,230	8.2	384	401
2,3,7,8	200	2.0	54	65
% 2,3,7,8 of total tetraCDF	16%	24%	14%	16%
PentaCDF	4,478	33	904	933
2,3,7,8 (2 isomers)	205	2.8	117	106
% 2,3,7,8 of total pentaCDF	4.6%	8.5%	13%	11%
HexaCDF	5,449	47	1,638	2,039
2,3,7,8 (4 isomers)	48	12	35	61
% 2,3,7,8 of total hexaCDF	0.9%	26%	2.1%	3.0%
HeptaCDF	2,044	23	942	977
2,3,7,8 (2 isomers)	695	8.7	354	342
% 2,3,7,8 of total HeptaCDF	34%	38%	38%	35%

1/ One sludge sample from the tank center; the second from tank corner.
2/ Reported by one laboratory, but not confirmed.

TABLE 7.7

CDD AND CDF CONGENER SPECIFIC ANALYSIS, PHASE 2: WOOD TREATMENT PLANT

(Summary of isomer group data and percent of group consisting of 2,3,7,8-chlorinated isomers, average of two laboratories except that, where differences exceed 5X, both values are reported.)

	"Bloom"	"Commercial"	Soil (Mouth of Pressurized Retort)	Sump (Liquid)
CDDs (ppb):				
TetraCDD	ND	ND	ND	ND[1/]
2,3,7,8	ND	ND	ND	ND
% 2,3,7,8 of total tetraCDD				
PentaCDD	70	ND	ND[1/]	ND[1/]
1,2,3,7,8	56	ND	ND	ND
% 2,3,7,8 of total pentaCDD	80%	-	-	-
HexaCDD	1,580	136	243	1,420 84
total 2,3,7,8 (3 isomers)	557	65 ND	96	414 54
% 2,3,7,8 of total hexaCDD	35%	51% 0	39%	29% 64%
HeptaCDD	26,680	9,530	2,420	12,900 548
1,2,3,4,6,7,8	23,570	6,650	1,522	8,270 343
% 2,3,7,8 of total HeptaCDD	88%	70%	63%	64% 63%
CDFs (ppb):				
TetraCDF	31	ND[1]	2.8	15
2,3,7,8	4.4	ND	ND	ND
% 2,3,7,8 of total tetraCDF	14%	-	0	0
PentaCDF	382	ND	43	484 27
2,3,7,8 (2 isomers)	56	ND	ND	50 13
% 2,3,7,8 of total pentaCDF	15%	-	-	10% 47%
HexaCDF	4,079	225	215	2,440 168
total 2,3,7,8 (4 isomers)	1,106	25	12	136 ND
% 2,3,7,8 of total hexaCDF	27%	11%	5.3%	5.6% 0
HeptaCDF	13,003	3,427	458 388	2,590 111
2,3,7,8 (2 isomers)	9,546	701	847 157	900 56
% 2,3,7,8 of total HeptaCDF	73%	20%	185% 40%	35% 50%

1/ Reported by one laboratory, but not confirmed.

laboratories for both isomer group and congener-specific studies. Results indicate that CDD and CDF residues in soil and dip tank sludges generally reflect the isomer group pattern seen for the products. The isomer group studies provided evidence of significant CDD and CDF levels associated with chlorophenol use. This established the need to determine the presence of specific congeners.

Congener-specific analysis was performed on additional samples in an effort to characterize the contribution of congeners chlorinated in the 2,3,7 and 8 positions to the isomer group totals. Samples obtained from three sawmills and one wood treatment plant included commercial chlorophenol products, dip tank liquid, dip tank sludge, a pressure treated wood "bloom", and soil, with samples split between contract laboratories for analysis.

As discussed elsewhere in this report, several investigators have reported levels of CDDs and CDFs in chlorinated phenols used for treatment of wood. However, with the exception of the 2,3,7,8-tetraCDD and 2,3,7,8-tetraCDF congeners, the levels are reported as isomer group data; levels of specific 2,3,7,8-chlorinated congeners of possible concern such as 1,2,3,7,8-pentaCDD have not been measured.

An exception is the recent work of Miles et al. (1985a,b) who synthesized the 10 hexaCDD isomers and used these standards to perform isomer-specific analysis of nine commercial pentachlorophenol samples (from three manufacturers) and six commercial sodium pentachlorophenate samples (from two manufacturers). The results of Miles et al. (1985b), summarized in Table 7.8, show that the specific isomer composition varies between pentachlorophenol and pentachlorophenate. Levels of total hexaCDD varied from 0.7 ppm to 38.5 ppm in the nine pentachlorophenol samples and from 1.6 ppm to 16.3 ppm in the pentachlorophenates. The predominant 2,3,7,8-hexaCDD isomer is 1,2,3,6,7,8 with a range of 50.6 percent to 64.5 percent of total hexaCDD in pentachlorophenols and 17.5 percent to 26.5 percent in pentachlorophenate. Much lower levels of the other two 2,3,7,8-hexaCDD isomers (1,2,3,4,7,8 and 1,2,3,7,8,9) were detected, although 1,2,3,7,8,9 was identified in all 15 samples.

The State Board's hexaCDD isomer-specific results agreed with Miles et al., that 1,2,3,6,7,8-hexaCDD was the predominant 2,3,7,8-hexaCDD (Table H.5 of Appendix H). The two commercial chlorophenate formulations contained 39 percent and 40 percent 1,2,3,6,7,8-hexaCDD of total hexaCDD. Dip tank sludges and liquids contained 50 percent to 58 percent 1,2,3,6,7,8-hexaCDD of the total hexaCDD group.

TABLE 7.8

PERCENTAGE OF 2,3,7,8-HEXACDD ISOMERS
IN TECHNICAL PCP AND ITS SODIUM SALT
(Adapted from Miles et al., 1985b)

PCP Sample	1,2,3,4,7,8,	1,2,3,6,7,8,	1,2,3,7,8,9,	Total Hexa-CDD	M*
1		50.6%	1.0%	38.5 ppm	A
2		52.2%	1.9%	36.8 ppm	A
3		56.8%	0.7%	37.5 ppm	A
4		58.9%	1.5%	0.7 ppm	B
5		60.1%	1.3%	1.9 ppm	B
6		62.8%	0.6%	1.4 ppm	B
7	0.6%	58.4%	1.0%	4.6 ppm	C
8	0.4%	62.5%	1.0%	2.8 ppm	C
9	1.1%	64.5%	1.0%	6.1 ppm	C
NA - PCP					
1	1.5%	19.7%	5.2%	15.4 ppm	D
2	1.5%	17.5%	5.2%	16.3 ppm	D
3	1.8%	19.0%	5.3%	14.8 ppm	D
4	1.4%	24.1%	2.8%	1.8 ppm	E
5	1.5%	25.2%	2.3%	1.6 ppm	E
6	1.7%	26.5%	2.4%	2.2 ppm	E

M* = Manufacturer

As shown in Table H.5 of Appendix H, the 2,3,7,8-chlorinated CDF congeners detected were 2,3,7,8-tetraCDF (approximately 15 percent of total isomer group in chlorinated phenol formulations), 1,2,3,7,8-pentaCDF, and 2,3,4,7,8-pentaCDF. The four 2,3,7,8-hexaCDF isomers represented only a small fraction (about one percent) of the total hexaCDFs. Reporting laboratories frequently disagreed on which specific 2,3,7,8 hexaCDF isomers were present.

At Sawmill A, the congener-specific CDD and CDF composition of the commercial formulation (sodium pentachlorophenate) used for treatment was compared to the dip tank sludge (see Table 7.5). Except for 1,2,3,7,8 pentaCDD, which was not detected in the sludge, the same 2,3,7,8 chlorinated congeners were present in both the formulation and the sludge. Concentrations of the CDDs and CDFs in the commercial formulation exceeded those in the sludge by roughly a factor of two. Because Sawmill A had been abandoned shortly before the samples were taken, no history of previous commercial formulations used for treatment was available. Thus, no direct comparison of sludge CDD and CDF content with that of chlorophenol treatment chemical(s) is possible.

At Sawmill C, concentrations in the commercial formulation (a potassium tetrachlorophenate) also exceeded the dip tank sludge by a factor of two (see Table 7.6). Again, no direct comparison is possible because the sludge had accumulated during the use of two different commercial formulations over a three and one-half year period. The concentration of CDDs and CDFs in the dip tank liquid were approximately one percent of those in the formulation, reflecting the one to one hundred dilution (formulation to water) used by the sawmill.

The highest CDD and CDF concentrations detected at the wood treatment plant were in the bloom, including 56 ppb 1,2,3,7,8-pentaCDD (see Table 7.7).

Based on the State Board's limited survey, Table 7.9 summarizes the tetra- through hexaCDD and CDF detected in chlorophenate formulations, dip tank sludges and a treatment plan "bloom".

TABLE 7.9

SUMMARY OF TETRA- THROUGH HEXA- 2,3,7,8-CHLORINATED CONGENERS DETECTED IN THE STATE BOARD SURVEY (CONCENTRATIONS IN ppb)

	Sawmill A		Sawmill C		
	Commercial Formulation	Dip Tank Sludge	Commercial Formulation	Dip Tank Sludge	Wood Treatmt Plant Bloom
CDDs					
2,3,7,8-tetraCDD	ND	ND	ND	ND	ND
1,2,3,7,8-pentaCDD	26	ND	ND	ND	56
2,3,7,8-hexaCDDs	3,825	1,795	509	225	557
CDFs					
2,3,7,8-tetraCDF	201	105	200	60	4.4
2,3,7,8-pentaCDFs	614	220	205	112	56
2,3,7,8-hexaCDFs	603	373	48	48	1,106

Table 7.10 compares levels of 2,3,7,8-chlorinated CDDs and CDFs detected in the twelve samples analyzed in the two phase congener specific program. Although 2,3,7,8-tetraCDD was confirmed in only one sample, 2,3,7,8-tetraCDF was detected in all of the sawmill samples and in the pressurized wood treatment "bloom". In 10 of 12 samples, 2,3,7,8-chlorinated CDD levels were higher than the CDFs. In four samples, the total level of tetra through hepta 2,3,7,8-chlorinated CDDs and CDFs exceeded 15,000 ppb: a commercial pentachlorophenate, two sawmill sludges, and the "bloom" taken from pressure treated wood. With the exception of the dip tank solution sample, all samples contained at least 1,000 ppb total tetra through hepta 2,3,7,8-chlorinated CDDs and CDFs.

This limited congener-specific survey has indicated the following tetra through hexa 2,3,7,8-chlorinated CDD and CDF congeners are most likely to be found as a result of tetrachlorophenol and pentachlorophenol use at sawmills and wood treatment plants:

1,2,3,6,7,8 hexaCDD
2,3,7,8 tetraCDF
1,2,3,7,8 pentaCDF
2,3,4,7,8 pentaCDF
1,2,3,6,7,8 hexaCDF

In addition, various other 2,3,7,8-chlorinated hexaCDFs were reported in some samples (in particular, these hexaCDFs were detected in the crystalline "bloom" that formed on pressure-treated wood).

TABLE 7.10

SUMMARY OF 2,3,7,8-SUBSTITUTED CDDs AND CDFs
IN TWELVE COMPOUND-SPECIFIC ANALYSES (TETRA, PENTA,
HEXA, AND HEPTA ISOMER GROUPS, CONCENTRATIONS GIVEN IN ppb)

Sample	2,3,7,8-Tetra CDD	2,3,7,8-Tetra CDF	Chlorinated CDDs	Chlorinated CDFs	Total 2,3,7,8 CDDs AND CDFs[1/]
Commercial Na-PCP, Sawmill A	0	201	34,751	6,540	41,291
Commercial K-TetraCP, Sawmill C	0	200	1,197	1,148	2,345
Sawmill Dip Tanks					
Sawmill A sludge	0	15	25,305	3,333	28,638
Sawmill B wet sludge	0	17	2,332	485	2,817
Sawmill B dry sludge	9.7	95	15,411	2,177	17,588
Sawmill C center sludge	0	54	1,092	560	1,652
Sawmill C corner sludge	0[2/]	65	1,161	574	1,735
Sawmill C liquid	0	2.0	18	26	44
Wood Treatment Plant- PCP "Bloom"	0	4.4	24,183	10,712	34,895
Recycled "Commercial"	0	0	6,715	726	7,441
Soil at Retort Mouth	0	0	1,618	169	1,887
Sump Liquid	0	0	8,684	69	8,753

1/ Does not include octaCDD and octaCDF.

2/ Reported at 6.8 ppb by one laboratory but not confirmed by second.

CHAPTER 8: HAZARD EVALUATION

Results of the State Board's program to perform congener-specific analyses for CDDs and CDFs present in samples taken at sawmills and a wood treatment plant have been presented in the previous chapter. Based on the study results, this chapter discusses approaches to evaluate CDD and CDF-contaminated sites.

Although 2,3,7,8-tetraCDD was confirmed in only one of twelve samples, other 2,3,7,8-congeners of toxicological concern were detected in all twelve. As a means to estimate potential risk, this chapter discusses three procedures to evaluate mixtures of CDDs and CDFs based on methods developed by the U. S. Environmental Protection Agency (Bellin and Barnes, 1986) and the California Department of Health Services (CARB and CDHS, 1986).

Various scenarios are summarized that evaluate the toxicity of CDD and CDF mixtures based on toxic equivalence relative to 2,3,7,8-tetraCDD. The State Board program included congener-specific analysis of 12 samples. The results indicated the proportion of 2,3,7,8-chlorinated isomers present within an isomer group. Based on this information, a simple procedure is suggested to estimate the concentration of 2,3,7,8-chlorinated CDDs and CDFs present in samples analyzed by less costly isomer group analysis.

Finally, the issue of determining cleanup levels for sites contaminated by mixtures of CDDs and CDFs is discussed. A site-specific approach that uses the California Site Mitigation Decision Tree Manual is suggested.

TOXIC EQUIVALENCY FACTORS

This section discusses three approaches to evaluate mixtures of CDDs and CDFs based on toxic equivalence to 2,3,7,8-tetraCDD. One approach has been developed by the U.S. EPA, and a second by the California Department of Health Services. A third procedure is simple summation of all tetra-through-heptaCDDs and CDFs.

U.S. EPA Approach

The U.S. EPA has developed an approach to assess the hazards presented by CDDs and CDFs in soot, incinerator fly ash, industrial waste, and soils (Bellin and Barnes, Interim Procedures for Estimating Risks Associated with Exposure to Mixtures of Chlorinated Dibenzo-p-Dioxins and Dibenzofurans [CDDs and CDFs], October 1986). EPA concluded that the preferred method to assess complex mixtures of CDDs and CDFs was by direct biological assessment. However, because the information for biological assessment of each 2,3,7,8-chlorinated congener is not currently available, the interim approach recommended has been

estimation of the risk potential of the most toxic CDDs and CDFs (i.e., 2,3,7,8-chlorinated congeners), by estimating their equivalence to 2,3,7,8-tetraCDD. EPA examined a range of experiments measuring the following systemic and biochemical effects:

- o cancer induction
- o reproductive effects
- o in vitro cell transformation
- o enzyme induction
- o receptor binding

EPA used the rationale listed below to establish the relative toxicity factors shown in Table 8.1.

1. Determination of toxicity factors for 2,3,7,8-tetraCDD and the 2,3,7,8-hexaCDDs was based on carcinogenic potency derived by the U.S. EPA's Cancer Assessment Group. Relative potency for the 2,3,7,8-hexaCDDs was four percent of 2,3,7,8-tetraCDD.

2. Relative toxicity for 1,2,3,7,8-pentaCDD was estimated to be 50 percent of 2,3,7,8-tetraCDD by using the arithmetic mean of carcinogenic potency values for 2,3,7,8-tetra- and hexaCDDs.

3. The 2,3,7,8-tetraCDF and 2,3,7,8-pentaCDFs were assigned a relative potency value of 0.1, based on in vitro and reproductive toxicity tests that showed these CDFs were one to two orders of magnitude less potent than 2,3,7,8-tetraCDD. Since in vitro tests show the hexaCDFs to be one tenth as potent as the pentaCDFs, the hexaCDFs were assigned a value of 0.01.

4. The heptaCDDs and heptaCDFs were assigned a value of 0.001 because their enzyme induction potency is about 0.001 of 2,3,7,8-tetraCDD.

5. EPA noted that, in most tests, the non-2,3,7,8 chlorinated CDDs and CDFs were one to three orders of magnitude less potent than their 2,3,7,8-chlorinated isomers. For non-2,3,7,8-chlorinated CDDs and CDFs, potency was set at 0.01 of the corresponding 2,3,7,8-chlorinated isomer(s). For example, 1,2,4,6,7,9-hexaCDD would have a toxic equivalency factor of 0.01 times the 0.04 value for a 2,3,7,8-hexaCDD, or 0.0004.

These estimates of relative potency can be refined if and when more information becomes available. Table 8.2 illustrates application of the U.S. EPA method to determine the toxic equivalency of the CDD and CDF mixture in a dip tank sludge that was analyzed as part of the State Board's congener-specific monitoring (Sawmill C). In this example, the CDFs contributed approximately twice as much relative toxicity concentration as

TABLE 8.1

CDD AND CDF CONGENERS OF MOST TOXIC CONCERN
(BELLIN AND BARNES, 1986)

	Congener	TEF[1/]
CDD		
	2,3,7,8-tetraCDD	1.0
	1,2,3,7,8-pentaCDD	0.5
	1,2,3,4,7,8-hexaCDD	0.04
	1,2,3,6,7,8-hexaCDD	0.04
	1,2,3,7,8,9-hexaCDD	0.04
	1,2,3,4,6,7,8-heptaCDD	0.001
CDF		
	2,3,7,8-tetraCDF	0.1
	1,2,3,7,8-pentaCDF	0.1
	2,3,4,7,8-pentaCDF	0.1
	1,2,3,4,7,8-hexaCDF	0.01
	1,2,3,6,7,8-hexaCDF	0.01
	1,2,3,7,8,9-hexaCDF	0.01
	2,3,4,6,7,8-hexaCDF	0.01
	1,2,3,4,6,7,8-heptaCDF	0.001
	1,2,3,4,7,8,9-heptaCDF	0.001

1/ TEF: Toxic Equivalency Factor: the estimated toxicity relative to 2,3,7,8-tetraCDD

TABLE 8.2

CALCULATION OF TOTAL RELATIVE TOXICITY CONCENTRATION
USING THE U.S. EPA METHOD:
CDD AND CDF CONCENTRATIONS MEASURED
IN SAWMILL C DIP TANK SLUDGE

Isomer Group	Concentration of 2,3,7,8 Congeners (ppb)	Toxic Equivalency Factor	Relative Toxicity Concentration (ppb)
CDDs			
TetraCDD	-	1.0	0
PentaCDD	-	0.5	0
HexaCDD	224	0.04	9.0
HeptaCDD	937	0.001	0.9
Total			9.9
CDFs			
TetraCDF	65	0.1	6.5
PentaCDF	106	0.1	10.6
HexaCDF	61	0.01	0.6
HeptaCDF	342	0.001	0.3
Total			18.0
Total Tetra-HeptaCDDs and CDFs			27.9

the CDDs. The estimated toxicity concentration related to 2,3,7,8-tetraCDD was 27.9 ppb. Using the U.S. EPA approach, the congeners contributing the most relative toxicity were the pentaCDFs (38 percent), the hexaCDDs (32 percent), and 2,3,7,8-tetraCDF (23 percent).

In September 1986, a U.S. EPA Science Advisory Board Subcommittee met to critique the Agency's interim approach of assessing toxicity of CDD and CDF mixtures by estimating the toxicity of individual CDD and CDF congeners relative to 2,3,7,8-tetraCDD. While the subcommittee made several suggestions to refine the method, the general conclusion was that the approach is a "successful interim attempt to articulate a scientific rationale and procedures for developing risk management decisions for mixtures which contain CDDs and CDFs related in structure and activity to 2,3,7,8-tetraCDD". The cover letter and text of the subcommittee's report are contained in Appendix J of this report.

California Department of Health Services Approach (CDHS Favored Scenario)

In Appendix B of "Health Effects of 2,3,7,8 Tetrachlorodibenzo-p-Dioxin and Related Compounds", the California Department of Health Services (CARB and CDHS, 1986) discussed four methods (scenarios) to estimate total potency of CDD and CDF mixtures. The first and most conservative scenario assumed that all CDDs and CDFs, including octaCDD and octaCDF, are as potent as 2,3,7,8-tetraCDD. Scenario 2 also assumed all CDDs and CDFs, except the hexaCDDs, octaCDD, and octaCDF, were equivalent to 2,3,7,8-tetraCDD. Based on carcinogenic potency, the hexaCDDs were rated as 3 percent as potent as 2,3,7,8-tetraCDD and the octaCDD and CDF were not rated. Scenario 3 was similar to the U.S. EPA method presented above, except that the hexaCDDs were assigned a potency of 0.03 instead of 0.04. Scenario 3 uses results of short-term tests in addition to carcinogenicity bioassays. Scenario 4, favored in the CDHS document, limits data examined to that provided by carcinogenicity bioassays. Since these bioassays have only tested 2,3,7,8-tetraCDD and a mixture of two 2,3,7,8-hexaCDD isomers, there are only two equivalence factors. In Scenario 4, CDHS assigns pentaCDD the same value as 2,3,7,8-tetraCDD and 1,2,3,4,6,7,8-heptaCDD the same value as the 2,3,7,8-hexaCDD isomers. The CDFs are given the same estimated potencies as the equivalent CDD isomer groups (pentaCDF equal to pentaCDD, hexaCDF equal to hexaCDD, etc.)

The CDHS document (CARB and CDHS, 1986) notes that the U.S. EPA approach is the least health conservative and requires the greatest number of assumptions about relative potency. Noting that most CDDs and CDFs have not been tested for long-term chronic toxicity, CDHS states that the use of short-term tests to estimate long-term effects (the method employed by the U.S. EPA) is "tenuous".

Table 8.3 shows calculation of estimated toxicity concentration by using CDHS's favored scenario for the same Sawmill C sludge sample determined earlier by the U.S. EPA method (Table 8.2). Whereas the U.S. EPA method totalled 27.9 ppb relative toxicity concentration, the CDHS favored scenario was 217.9 ppb. The CDFs accounted for 84 percent of the relative toxicity, with pentaCDFs accounting for 49 percent (106 ppb) and 2,3,7,8-tetraCDF for 30 percent (65 ppb). The heptaCDD dominated the CDD contribution, with 13 percent (28.1 ppb) of the total and exceeded the 2,3,7,8-hexaCDD isomers by a factor of 4.

Summation of All Tetra-Through-HeptaCDDs and CDFs Approach

This is the simplest of the three approaches: all 2,3,7,8-tetra-, penta-, hexa-, and heptaCDDs and CDFs are added together. All these congeners are assigned the same toxic equivalency factor of 1.0. This approach (also shown in Table 8.3) yielded a concentration of 1,735 ppb in the dip tank sludge with the CDDs accounting for 67 percent of the total CDDs and CDFs.

Comparison of Toxic Equivalency Approaches

Using the State Board's results from all 12 samples that underwent congener-specific analysis, Table 8.4 summarizes three approaches to determine toxic equivalency of CDD and CDF mixtures to 2,3,7,8-tetraCDD. The three approaches are:

- U.S. EPA interim method
- CDHS Favored Scenario
- Total tetra- through heptaCDDs and CDFs

Use of the CDHS method resulted in higher estimated relative toxicity concentration. For the eight sawmill samples, CDHS estimates were four to nine times those of U.S. EPA. For the wood treatment plant, CDHS values were 11 to 28 times those of U.S. EPA's. Because the hexa and hepta isomer groups are given equal weight under the CDHS system of ranking, the presence of heptaCDDs and CDFs assumes more importance than with the U.S. EPA approach.

The method of adding all tetra- through hepta- 2,3,7,8 congeners provided much higher estimates of total relative toxicity concentration. Since this method treats all 2,3,7,8 chlorinated CDDs and CDFs as equal, very high estimates are given. The highest value, from the drum of commercial formulation at Sawmill A, totalled 41,291 ppb for total tetra- through heptaCDDs and CDFs. While the most health conservative, this approach excludes the results of long- and short-term studies that ranked CDDs and CDFs by relative potency.

TABLE 8.3

CALCULATION OF TOTAL RELATIVE TOXICITY CONCENTRATION
USING THE CDHS APPROACH:
CDD AND CDF CONCENTRATIONS MEASURED
IN SAWMILL C DIP TANK SLUDGE

Isomer Group	Concentration of 2,3,7,8 Congeners (ppb)	CDHS Toxic Equivalency Factor	Relative Toxicity Concentration (ppb)
CDDs			
TetraCDD	-	1.0	0
PentaCDD	-	1.0	0
HexaCDD	224	0.03	6.7
HeptaCDD	937	0.03	28.1
Total CDDs	1,161		34.8
CDFs			
TetraCDF	65	1.0	65
PentaCDF	106	1.0	106
HexaCDF	61	0.03	1.8
HeptaCDF	342	0.03	10.3
Total CDFs	574		183.1
Total Tetra-HeptaCDDs and CDFs	1,735		217.9

TABLE 8.4

HAZARD EVALUATION: TOTAL RELATIVE TOXICITY CONCENTRATIONS (ppb) OF 2,3,7,8 CHLORINATED CDDs AND CDFs USING THREE METHODS OF CALCULATION

SAMPLE	U.S. EPA (1986)[1/]	CDHS 1986[2/]	SUM OF ALL CDDs AND CDFs[3/]
Commercial Na-PCP			
Sawmill A	289.5	2,055	41,291
Commercial K-tetraCP			
Sawmill C	72.8	463	2,345
Sawmill Dip Tanks			
Sawmill A sludge	139.1	1,184	28,638
Sawmill B wet sludge	32.0	173	2,817
Sawmill B dry sludge	329.6	1,094	17,588
Sawmill C center of tank sludge	27.0	216	1,652
Sawmill C corner of tank sludge	27.9	218	1,735
Sawmill C liquid	0.8	8.4	44
Wood Treatment Plant-PCP			
"Bloom"	100.5	1,120	34,895
Recycled "Commercial"	11.3	223	7,441
Soil at Retort Mouth	5.6	64	1,887
Sump Liquid	9.8	274	8,753

1/ Bellin, J. and D. Barnes. 1986. Interim Procedures for Estimat Risks Associated with Exposure to Mixtures of Chlorinated Dibenzodioxins and Dibenzofurans

2/ CDHS, 1986; Favored Scenario where relative potency of 2,3,7,8-tetra- and pentaCDDs and CDFs = 2,3,7,8-tetraCDD and 2,3,7,8-hexa- and heptaCDDs and CDFs = 2,3,7,8 hexaCDD (or 0.03 2,3,7,8-tetraCDD).

3/ Excluding octaCDD and octaCDF.

ESTIMATION OF 2,3,7,8 CONGENER-SPECIFIC CONCENTRATIONS FROM ISOMER GROUP DATA

Because of the complexity and cost associated with 2,3,7,8 congener-specific analysis, most analyses of CDD and CDF mixtures have been reported in terms of isomer groups. Unfortunately, isomer group data do not provide an indication of concentrations of the most toxic congeners of concern. Results from the limited State Board study can be adapted to estimate the concentrations of 2,3,7,8 congeners present in analyses reported as isomer group data.

In previous studies, two simple alternative procedures have been used (CARB and CDHS, 1986):

1. Assume all isomers in an isomer group are chlorinated at the 2,3,7, and 8 positions. For example, if 100 ppb hexaCDD is reported in an analysis, the level of 2,3,7,8-hexaCDDs would be 100 ppb.

2. Assume each isomer in an isomer group has an equal chance of occurrence. Since there are ten possible hexaCDD isomers and three are chlorinated at the 2,3,7, and 8 positions, then the estimated amount of 2,3,7,8-hexaCDDs would be (3/10) (100 ppb) = 30 ppb.

In contrast, the State Board has conducted a limited program (12 samples) of congener-specific analyses. This allows for inferences to be made as to approximate percentages of 2,3,7,8-congeners present, based on measured concentrations instead of assumptions. Table 8.5 is a condensation of Table H.5 in Appendix H, "Results of State Board CDD and CDF 2,3,7,8 Congener-Specific Analyses", and the percentages given are based on this very limited number of analyses. For example (referring to Table 6.5), if an isomer group analysis of sawmill dip tank sludge shows 100 ppb hexaCDD, then the estimated 2,3,7,8-chlorinated hexaCDD level will be (100 ppb) (55%) = 55 ppb.

In performing a hazard evaluation, either the U.S. EPA or CDHS approach can be used after converting the results of isomer group analysis to calculated 2,3,7,8 congener-specific concentration. Analysis by isomer group of CDDs and CDFs has the advantage that it can be performed currently by many laboratories at less cost and more rapidly than congener-specific analysis, which only a few laboratories are capable of attempting. Further, if a discharger disagrees with the estimated percentage of 2,3,7,8 congeners, he can arrange for congener-specific analysis to support his argument.

TABLE 8.5

APPROXIMATE PERCENTAGE OF 2,3,7,8-CHLORINATED ISOMERS DETECTED IN EACH CDD AND CDF ISOMER GROUP

Isomer Group	Commercial Na-PCP	Commercial K-tetraCP	Dip Tank Sludge	Wood Treatment Plant-Bloom & Soil
CDDs				
TetraCDD (1 of 22)[1/]	ND	ND	ND	ND
PentaCDD (1 of 14)	12%	ND	ND	80%
HexaCDD (3 of 10)	39%	40%	55%	45%
HeptaCDD (1 of 2)	44%	64%	60%	70%
CDFs				
TetraCDF (1 of 38)	14%	16%	19%	14%
PentaCDF (2 of 28)	7%	5%	10%	14%
HexaCDF (4 of 16)	2%	1%	4%	20%
HeptaCDF (2 of 4)	10%	34%	34%	43%

1/ Numbers in parentheses indicate the number of 2,3,7,8-chlorinated isomers possible within an isomer group. For example: HexaCDD (3 of 10) indicates three of ten isomers are chlorinated at 2,3,7, and 8 positions.

SETTING A CLEANUP LEVEL

The U.S. EPA established a site-specific cleanup level of 1 ppb 2,3,7,8-tetraCDD for the town of Times Beach, Missouri. The level was recommended to U.S. EPA in an extensive analysis performed by the Centers for Disease Control (Kimbrough et al., 1984) and is based on reasonable human risk of exposure. Kimbrough et al. (1984) suggest that 2,3,7,8-tetraCDD at levels of 1 ppb or greater in residential soil is of concern and "cannot be considered safe". A level set for industrial locations, such as a sawmill or wood treatment plant, possibly may exceed that for residential soil.

In Table 8.4, the twelve samples analyzed by congener-specific analysis were examined for toxic equivalency to 2,3,7,8-tetraCDD. Using the U.S. EPA method, only one sample--the dip tank liquid at Sawmill C at 0.8 ppb--had a toxic equivalency factor of less than 1 ppb. With the CDHS method, all twelve exceed 1 ppb. It should be noted that the 1 ppb level was determined for residential exposure, a scenario that includes potential ingestion of contaminated soil by young children. Most sawmill and wood treatment plant sites examined during the State Board investigation do not fit a residential exposure scenario.

The Times Beach clean-up level of 1 ppb was site-specific and should not be used arbitrarily at other sites. Rather, each site found to contain CDDs and CDFs will require independent evaluation. If these compounds are detected in soils sampled at an abandoned sawmill, then the potential for human contact or environmental contamination needs to be determined. In California, The California Site Mitigation Decision Tree Manual (Decision Tree) (CDHS, 1986) has been developed by the State Department of Health Services to provide a basis for risk management decisions. The Decision Tree includes five components: (1) preliminary risk appraisal, (2) site assessment, (3) risk appraisal, (4) environmental fate and risk determination, and (5) determination of mitigation strategy and remedial action plan selection. A Decision Tree approach to cleanup of CDD and CDF-contaminated soils at the abandoned sawmill will require a number of important site-specific evaluations during site assessment. Field observations are required to identify the contaminants, the exposure pathways (air, soil, water, biota), and the biological receptors (e.g., humans, aquatic species).

Based on site-specific field observations, the Decision Tree process proceeds to risk appraisal. During risk appraisal, statewide criteria are established with Applied Action Levels (AALs). The AALs are set for each medium of exposure (air, soil, water, biota) to protect specific biological receptors. The Decision Tree defines an applied action level as "a criterion which delineates a concentration of a substance in a media which when exceeded is determined to present a significant risk of an

adverse impact to a biological receptor" (CDHS, 1986). The Department of Health Services is currently developing AALs for mixtures of CDDs and CDFs. The AALs are derived from maximum exposure levels at which no adverse effects appear in the biological receptor and are given in units of concentration. (An AAL for water would be expressed as mg/L; an AAL for soil as mg/kg.)

Derivation of AALs also takes into account the amount of medium taken in by inhalation, ingestion, and absorption as well as toxicokinetic factors (e.g., absorption, metabolism, distribution, and elimination) characteristic of the medium. For non-threshold agents (such as CDDs and CDFs), the AAL derivation is based on exposure at a level where risk is no greater than one in one million.

If an AAL is exceeded in any medium of exposure (soil, water, air, biota), and a significant risk identified, a risk management process should be identified that will mitigate the potential exposure. For further explanation, the Executive Summary to the CDHS Decision Tree is reproduced in Appendix J.

For the sake of illustration, the site-specific level of 1 ppb 2,3,7,8-tetraCDD set for Times Beach, Missouri can be applied to a hypothetical site in California. An abandoned sawmill contains CDDs and CDFs in surface soils and is adjacent to a residential area. Without mitigation measures, young children potentially will play in and ingest the contaminated soils. For this site, with potential exposure to children, the AAL (soil) will be 1 ug/kg (1 ppb). If analysis for CDDs and CDFs in the soil surface layer is performed, and the calculated toxic equivalency factor to 2,3,7,8-tetraCDD equals or exceeds 1 ppb, then remedial action will be required. The key to the Decision Tree approach is determination of the exposure pathway and potential exposure of a specific population. Because studies have shown that children five years old or younger ingest the highest amounts of soil, this age would compose the highest risk group. If only adults were potentially exposed, the AAL (soil) probably would be higher. And, if the contaminated soil was buried and the situation determined to be sufficiently stable that there would be no migration of CDDs and CDFs, then the Decision Tree would not require remedial action.

In summary, evaluation of a site containing mixtures of CDDs and CDFs should be site-specific before the necessity for cleanup can be determined. Characterization of the CDD/CDF mixture by toxic equivalence to 2,3,7,8-tetraCDD will allow an estimate of potential hazard. Site observations will determine if these compounds are likely to migrate (e.g., presence of solvents as co-contaminants, depth to groundwater, nearness to surface, etc.) or are essentially immobile. The pathways of potential exposure (air, soil, water, biota) can be established from site

evaluation. Potential biological receptors (aquatic life, occupationally-exposed workers, young children) to the CDD/CDF mixture need to be determined as part of the risk appraisal.

By using a thorough evaluation based on the Decision Tree approach, the options for remedial action can be identified. At some sites, moving the material may create more of a hazard than encapsulation and on-site storage. On-site storage, with material isolated from man and the environment, may be the most effective interim measure until acceptable methods of CDD/CDF destruction are available.

CALIFORNIA SITE MITIGATION DECISION TREE MANUAL

EXECUTIVE SUMMARY

Introduction

The purpose of the California Site Mitigation Decision Tree is to provide State decision makers with a standardized approach to setting site-specific mitigation criteria. The Decision Tree will also facilitate evaluation of remedial action alternatives to select the best plan based on scientific considerations of factors relating to public health and the environment while considering demographic factors, local concerns, and other variables.

Major elements contained within the Decision Tree include processes for setting Applied Action Levels (AALs) for contaminants in soil, water, air and biota in an expeditious manner; identifying specific data to be gathered; identifying preferred data gathering techniques and developing site mitigation criteria for alternative remedial actions.

It should be noted that the Decision Tree process establishes both Applied Action Levels (AALs) and site mitigation criteria. AALs are exposure criteria applied to all sites throughout the State. AALs delineate concentrations of toxic substances that, when exceeded, place a specified biological receptor at significant risk. Because AALs are biological receptor specific, not site specific, they have statewide applicability. The mitigation criteria, on the other hand, are site-specific criteria that a remedial action must achieve to keep the exposure level at the biological receptor below the AAL (i.e., below significant risk).

The Decision Tree process consists of five components. These components include Preliminary Site Appraisal, Site Assessment, Risk Appraisal, Environmental Fate and Risk Determination, and Determination of Mitigation Strategy and Remedial Action Plan Selection. The relationship between these components and an overview of the Decision Tree process are shown in Figure 1.

The Decision Tree process was designed to be applied to a variety of sites that may range from small, relatively simple sites to large, highly complex and difficult sites. Because of this diversity, the Decision Tree document is rather massive. However, its size should not intimidate the user. If the site is small and relatively easy to mitigate, many of the decision branches are never opened and the decision process is very rapid. If the site is complex, many of the decision branches must be opened and pursued. In either case, the decision points and the data requirements needed to make sound decisions are defined.

COMPONENT I: Preliminary Site Appraisal

Sites in California contaminated with hazardous wastes have been identified by regulatory agencies such as the State of California Department of Health Services, the California State Water Resources Control Board and the nine Regional Water Quality Control Boards, the United States Environmental Protection Agency, and a myriad of local agencies responsible property owners reporting contamination problems; and concerned citizens, including residents and past and present employees of companies that have contaminated sites. Preliminary Site Appraisal (PSA) is initiated by the discovery of a site which is potentially contaminated with hazardous substances. Based on the

characteristics of the wastes present and the features of the site itself, the site may be determined to be sufficiently hazardous to be placed on either the National Priority List (NPL) and/or the State Bond Act Expenditure Plan (CSBAEP).

The process to rank sites on the NPL and CSBAEP is primarily based on qualitative information, and does not include the detailed investigation required to fully characterize a site. Therefore, additional information may be required to implement subsequent components of the Decision Tree process. The Decision Tree process may also be used for sites that are not listed on the NPL and CSBAEP.

The PSA component of the Decision Tree identifies the universe of contaminants potentially present at the site using information acquired from the NPL or CSBAEP process. Additional investigation may be needed regarding the use of chemicals, production of wastes, and disposal practices at the site. The universe of contaminants is verified by a limited sampling program of the areas where contamination is most likely to be found.

An initial investigation of the adverse effects of the contaminants is necessary to determine the potential endangerment to human health. Determination of the likelihood of adverse effects occurring upon exposure to the contaminants is based upon available toxicologic data. Available data may show that adverse human health effects are imminent, and appropriate emergency action is necessary to protect public health.

Determination of the occurrence of health effects due to low level, chronic exposure is more difficult and requires additional investigation of the extent and magnitude of contamination, and the toxicologic properties of the contaminants, individually and collectively. Once it has been established that additional site assessment is necessary to provide data to develop appropriate remedial measures, the remaining components of the Decision Tree are initiated.

COMPONENT II: Site Assessment

The second component of the Decision Tree includes a preliminary evaluation of the site specific factors that affect the tendency of a hazardous substance to move between environmental compartments (air, soil, water, and biota). First, critical exposure pathways are identified. These pathways are the means by which exposure to contaminants in air, soil, water and biota occurs. Water pathways include both surface and ground waters.

In the first phase of identifying critical exposure pathways, determination of the current contaminant concentrations at points of exposure to biological receptors of concern should be made. The measurements or estimates made at this point are not thought of as static; concentrations change with time. Hazardous substances are not assumed to be isolated from biological receptors in separate environmental compartments. The Decision Tree process identifies data to be gathered for each pathway and cites preferred methods of data collection.

Typically, in this initial stage, samples are taken of waste, surface soils and shallow soils, runoff and surface water and ground water from existing wells. Sampling data from these site assessment activities will be the basis for a decision as to whether expanded air, soils, and ground water investigations will be necessary.

Expanded Assessment

If an expanded assessment of the site is warranted, evaluation of the environmental setting as it affects the behavior of the contaminants is required. The quantification of the meteorological, biological, soils and hydrologic systems at the site, together with information about the chemical and physical properties of the contaminants, forms a basis to evaluate the environmental fate of contaminants.

The collection of the necessary and sufficient data to adequately characterize the site and contaminants is the principal objective of this component. Site investigation programs are iterative in nature and will often require subsequent sampling and monitoring device installation to resolve issues that arise after data collected during initial site appraisal are analyzed. Although environmental compartments are presented as separate modules, contaminant transfer occurs across compartment boundaries and processes in one compartment often influence processes in another.

Quality Assurance/Quality Control

As the process is described in the Decision Tree Manual, data obtained from field sampling and analysis and/or literature values may be used to determine existing and future concentrations of chemical contaminants in environmental media (i.e., air, soil, water, and biota). Regardless of the source, it is important that these data be accurate, precise, complete, representative and comparable to other appropriate data.

To ensure that all data used in the process described in this manual are representative of environmental conditions, the Quality Assurance/Quality Control Plans (QA/QC) used in the data generation need to be evaluated. The main components of a QA/QC plan that need special scrutiny are the basis for measurement, experimental information, statistical information (e.g., means, ranges, and standard deviation), and corroborative information.

The QA/QC plans for sampling and for analysis should be developed together. At a minimum, the data generators and users should work together in developing an integrated site-specific QA/QC plan.

COMPONENT III: Risk Appraisal

An evaluation of the effects produced by toxic substances which originate from waste sites centers on appraising the adverse impacts of these substances on the public health and the surrounding ecosystem. Every potential effect is not, and should not be, delineated by the appraisal process. Given the limited resources that are available and the complexity of the numerous sites scattered throughout California, the appraisal must focus on those biological receptors of concern that are potentially at risk. The Decision Tree process is aimed at ensuring their protection.

Three types of information are essential to evaluate the sites.

1. The toxic substances are identified from data collected in the Site Assessment Process.

2. The biological receptors of concern in the ecosystem potentially impacted by the toxic substances are identified in the Site Assessment Process.

3. The critical exposure pathways are delineated in the Site Assessment Process.

A criterion will be identified or developed for maximum acceptable exposure for toxic contaminants. The criteria are employed to identify significant adverse effects of the toxic contaminants on the biological receptors. These criteria, denoted as Applied Action Levels, are applicable statewide. An Applied Action Level (AAL) is specific to a toxic substance, a biological receptor and a medium of exposure.

The methodology employed to develop AALs is quite conventional. It is a compilation of the approaches outlined by the U.S. Environmental Protection Agency, the National Academy of Sciences and the California Department of Health Services. Toxic Substances are grouped into two catagories for the purpose of developing AALs. For carcinogens, mutagens and genotoxic teratogens no threshold for an adverse effect is assumed. The AALs are based on a maximum exposure level (MEL) which produces one adverse effect in a population of one million exposed. The MELs are determined from epidemiological research or long-term animal bioassays.

For other toxic agents a threshold for an adverse effect is assumed. The AAL is established, with a margin of safety, at the maximum exposure level which does not produce an adverse effect. Uncertainties associated with the above

approaches are factored into the criteria to ensure a margin of protection for the biological receptors of concern.

The total exposure of biological receptors to toxic substances via various media is evaluated at their site(s) of exposure. The fractions of the AAL present in each media of exposure are added. When the total cumulative exposure exceeds an MEL, a significant risk to a biological receptor is indicated and a risk management process is warranted.

Exposure to substances that produce the same toxic manifestation or are considered likely to interact is also appraised. When the total cumulative exposures to toxic chemicals in all media of exposure constitute a significant risk to a biological receptor, the initiation of a risk management process is warranted.

Whenever an Applied Action Level is exceeded in any media of exposure, an assessment will be made to determine the necessity for interim actions to be implemented immediately to protect public health and the environment. A few examples of immediate interim actions are: fencing the contaminated site, covering exposed contaminated soils, and restricting use of water. Immediate interim actions will usually not be the ultimate containment or treatment strategy. Interim actions are developed primarily to reduce public exposure prior to initiating the final Remedial Action Plan (RAP).

COMPONENT IV: Environmental Fate and Risk Determination

Environmental Fate - Subsurface Conditions, Soils and Groundwater

In producing this section of the Decision Tree Manual, it is recognized that diverse subsurface conditions are encountered in hazardous waste site investigations and that considerable flexibility and professional judgement are often required to conduct an investigation of subsurface geology, hydrology, and soil and ground water contamination. Items to be considered include those factors that could act to transfer contaminants adsorbed to soil particles through the soil column. Factors of concern are: infiltration of precipitation, leakage of liquids from underground storage or conveyance structures, and spillage or other discharges to ground that could encourage leaching of contaminants from soil. In many cases, both current and future land use must be considered to evaluate the effect of environmental factors on the contaminants residing in the soil column.

Patterns of soil contamination existing at a hazardous waste site may often be the result of waste disposal events that took place over many years. Variations in the waste type, climate and precipitation, micro-structure of the soils, biological activity, and soil-chemical interactions can act to result in a complex pattern of soil contamination. Evaluation of patterns of soil contamination at a hazardous waste site should begin with recognition of any qualitative similarities or discernible trends that might be corrected with stratigraphy. Initial perceptions should be validated by actual field sampling.

The ground water investigation, including water quality and hydrological assessment, should occur in coordination with the subsurface soils investigation.

The hydrological portion of the investigation should start with a description of regional and site-specific ground water hydrology. This will include the identification of recharge and discharge areas and rates, presentation of regional and site-specific potentiometric surface contours, estimates of aquifer properties and parameters, and description of the hydrological relationship between zones of concern.

The investigation of water quality condition should include identification and description of plumes of contamination, extent of contamination with reference to known sources of contaminants and direction of ground water flow, vertical stratification of contamination, water quality of upgradient wells, estimates of the rate of movement of contaminants, and potential for contamination of downgradient wells.

Ground water systems are most often complicated and heterogeneous in nature. Distribution and transport analyses should, therefore, be done with an understanding of the inherent limitation and approximations.

Environmental Fate - Biota

The process leading to an accumulation of chemical residues in the body of an organism, above the levels in the environment or food, is termed bioconcentration. Accumulation of chemical residues can occur by direct adsorption from water or air as well as by ingestion. Bioconcentration of chemicals by nonhuman organisms (aquatic or terrestrial plants and animals) is of interest because of the potential for human exposure through consumption of these organisms and the potential direct impact of the chemicals on the accumulating organism. Bioconcentration is a public health concern with food items that may have potentially harmful chemical residues, and thereby pose a risk to the consumer. Methods for estimating or measuring bioconcentration are presented in the Decision Tree Manual.

Environmental Fate - Air

There is increasing evidence that air emissions of toxic chemicals from hazardous waste sites may pose a health threat to persons who live, play or work in the vicinity of the site. Volatile chemicals may be released as gases from a variety of sources including landfills, surface impoundments (also called ponds or lagoons), contaminated land or surface waters, land treatment areas, deteriorating containers or tanks. Chemicals adsorbed to soil may also be transported as windblown particulate matter, especially in areas with frequent vehicular or mechanical disturbance.

The Decision Tree Process provides an analytic framework for assessing the magnitude of existing or potential air contamination which may arise from hazardous waste sites. A complete investigation involves preliminary screening based on chemical and site characterstics followed by the use of calculations and field monitoring approaches to estimate emission rates and air concentrations of toxic chemicals.

The most important chemical parameters to consider when evaluating air emissions from a hazardous waste site are the vapor pressure and the Henry's Law

Constant. Vapor pressure, defined as the pressure exerted by a gas when in equilibrium with the liquid or solid phase, is a useful screening indicator of the potential of a chemical to volatilize from land. The Henry's Law Constant, which describes equilibrium partitioning of a chemical between solution in water and the gas phase of the chemical, is the most relevant parameter to estimate the tendency of a chemical to volatilize from a surface impoundment or water.

For wastes which have been deposited in landfills, mixed in the ground, or have seeped downward from soil surface contamination, Henry's Law Constant, indicates the tendency of the chemical to partition between soil water in the vadose zone or ground water and the soil vapor phase. This partitioning is the first step for volatile air emissions from hazardous wastes beneath the soil surface. The vapor pressure and Henry's Law Constant, however, are not sufficient to provide a good indication of the magnitude of an air emission problem from volatile chemicals. Site-specific characteristics must be considered.

The environmental characterstics of the site are a major factor influencing the potential for, and extent of, an air emission problem. Soil characteristics such as porosity, moisture, and organic content are particularly significant when evaluating volatile emissions from land. Adsorption of a chemical to the soil reduces the extent of volatilization. Precipitation and downward movement also decrese the concentration of the chemical which will reach the air. Meteorologic conditions such as temperature, wind speed, and barometric pressure may influence emission rate from waste sites; other meteorologic characteristics such as wind speed and direction influence the movement of the chemical once it is released and, ultimately, the concentration at biological receptors.

Analytic techniques selected for inclusion in this manual were based upon the accuracy of description of the phenomena and availability of input data. The selected emission rate estimation methods for various types of hazardous waste sites include most of the methods selected by the Environmental Protection Agency. These approaches generally provide conservative estimates of downwind conditions that would not be expected to be exceeded.

Risk Determination

The appraisal of the adverse impacts of toxic substances on biological receptors at concentrations predicted to occur in the future is essentially identical to that employed to evaluate the adverse impacts of existing concentrations of toxic substances. Once a predicted level of contamination is determined, the AALs are employed to appraise the risks associated with the predicted levels of exposure. Should a significant risk be identified, a risk management process should be initiated.

COMPONENT V: Development of Mitigation Strategies and Remedial Action Selection

The Decision Tree Process comes to conclusion in this fifth component. Based on the degree of hazard and the characterstics of the site, alternatives for

remedial action can be identified. It is anticipated that the alternatives will be developed either by the responsible party or by regional contractors working for the State. When appropriate, State staff will develop a preferred alternative.

The objective of site mitigation is to assure that the biological receptors associated with each environmental pathway are not exposed to hazardous chemcials at levels above the Applied Action Levels (AALs). The strategies developed to achieve this objective may include control of the pathway (such as ground water extraction and treatment), modification of the pathway (such as capping a site to reduce infiltration), or control of the source material (such as on-site stabilization or treatment of contaminated soils). The physical, legal, and administrative actions necessary to implement site mitigation and maintain the desired effects of the site mitigation strategy are developed in the Remedial Action Plan (RAP). State staff and regional contractors will evaluate likely remedial alternatives.

Selection of the preferred remedial action should be made based on the scientific and technical evaluations cited in the preceding text. However, local, political, social, and other considerations must also be factored into the final decision. For instance, if a small site of marginal threat to health and environment exists within a widely contaminated industrial setting, the decision makers must consider if the public's best interests are being served through the implementation of an extensive remedial action. Factors that need to be addressed include the availability or unavailability of resources to mitigate other sources of contamination that are of comparable or greater significance, costs of materials and required manpower, the costs of transportation and disposal if soil or water removal is an option, and exposures likely to occur resulting from uncovering buried wastes.

The utilization of the Decision Tree Process in development of site mitigation strategies and RAPs will include a re-evaluation of the site to determine if post-mitigation exposure to hazardous chemicals associated with a particular site will exceed AALs.

CONCLUSION

The California Site Mitigation Decision Tree provides a systematic method for identifying and evaluating the risk associated with abandoned or uncontrolled hazardous waste sites. Because of the rapidly changing nature of scientific and technical knowledge in this area, the Decision Tree process has been designed for flexibility and expandability.

The Decision Tree process provides State decision makers with a logical, systematic, and time efficient approach to mitigating contaminated sites. This represents significant progress in meeting the challenge of protecting the public health and the environment from adverse effects of exposure to toxic chemicals found on these contaminated sites.

I. PRELIMINARY SITE APPRAISAL

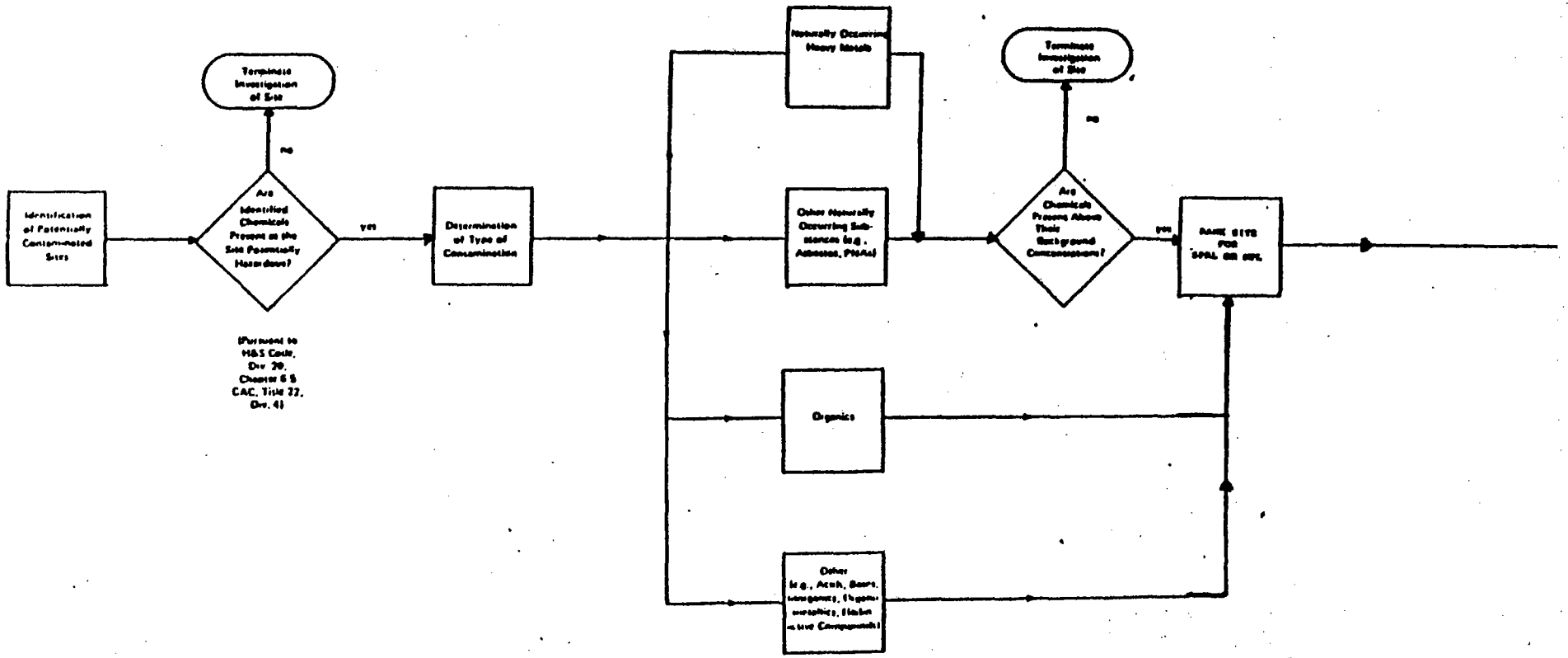
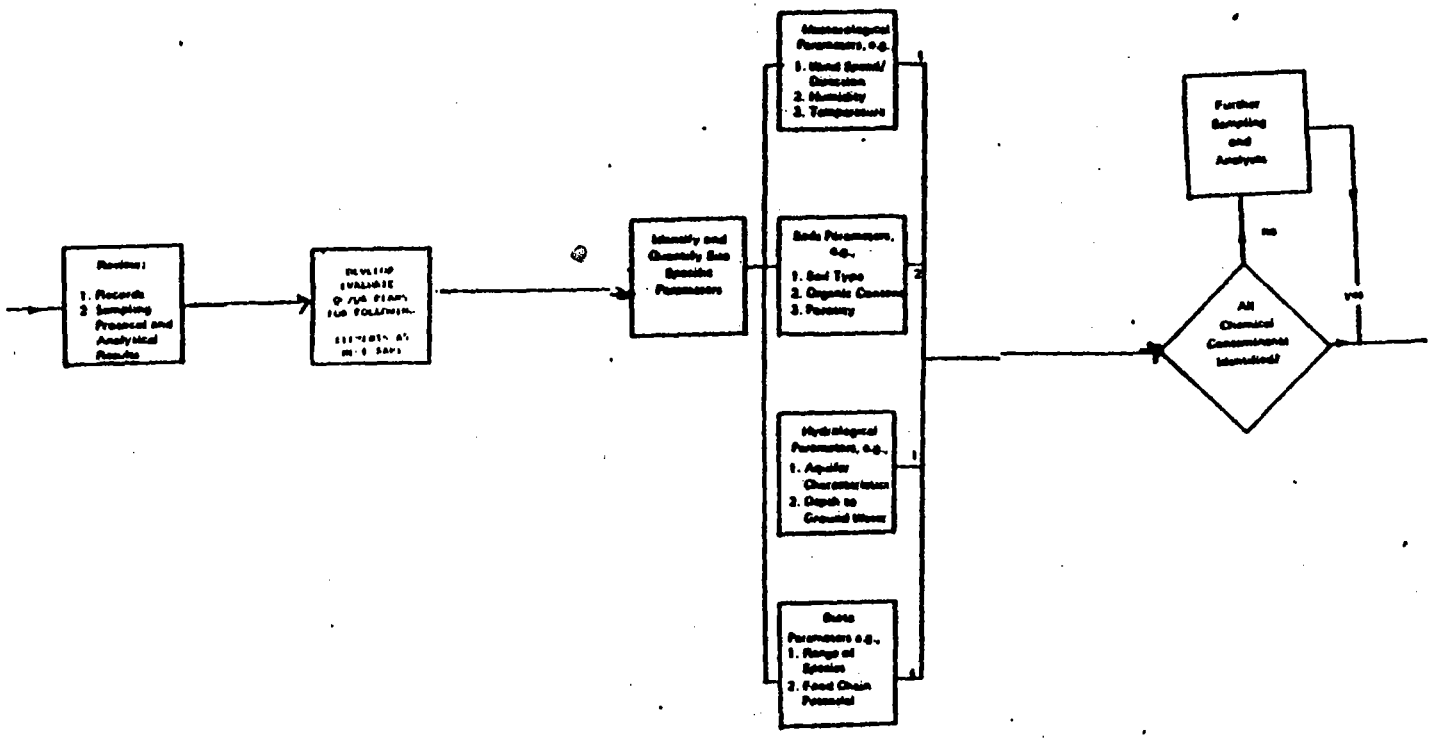
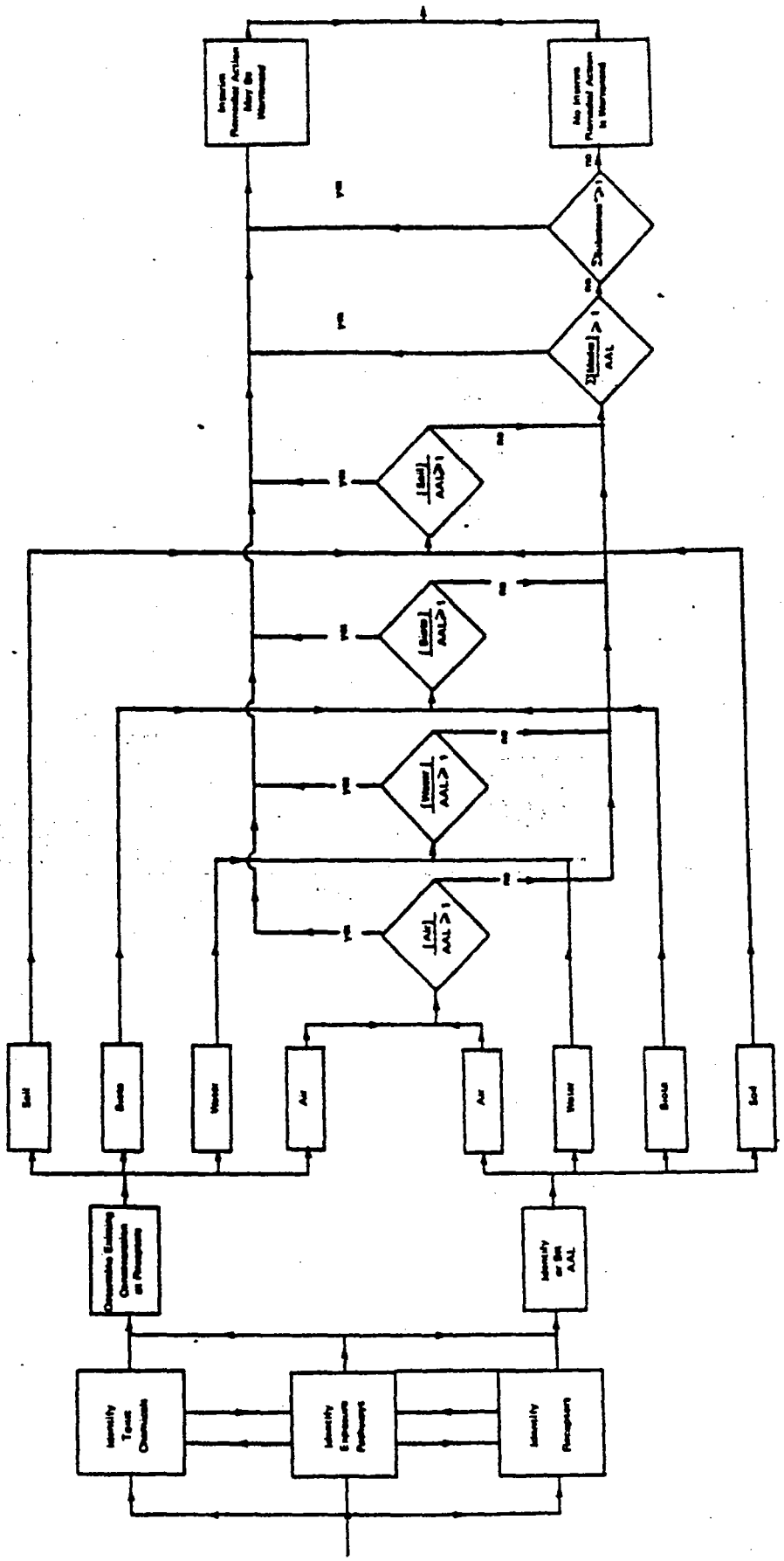
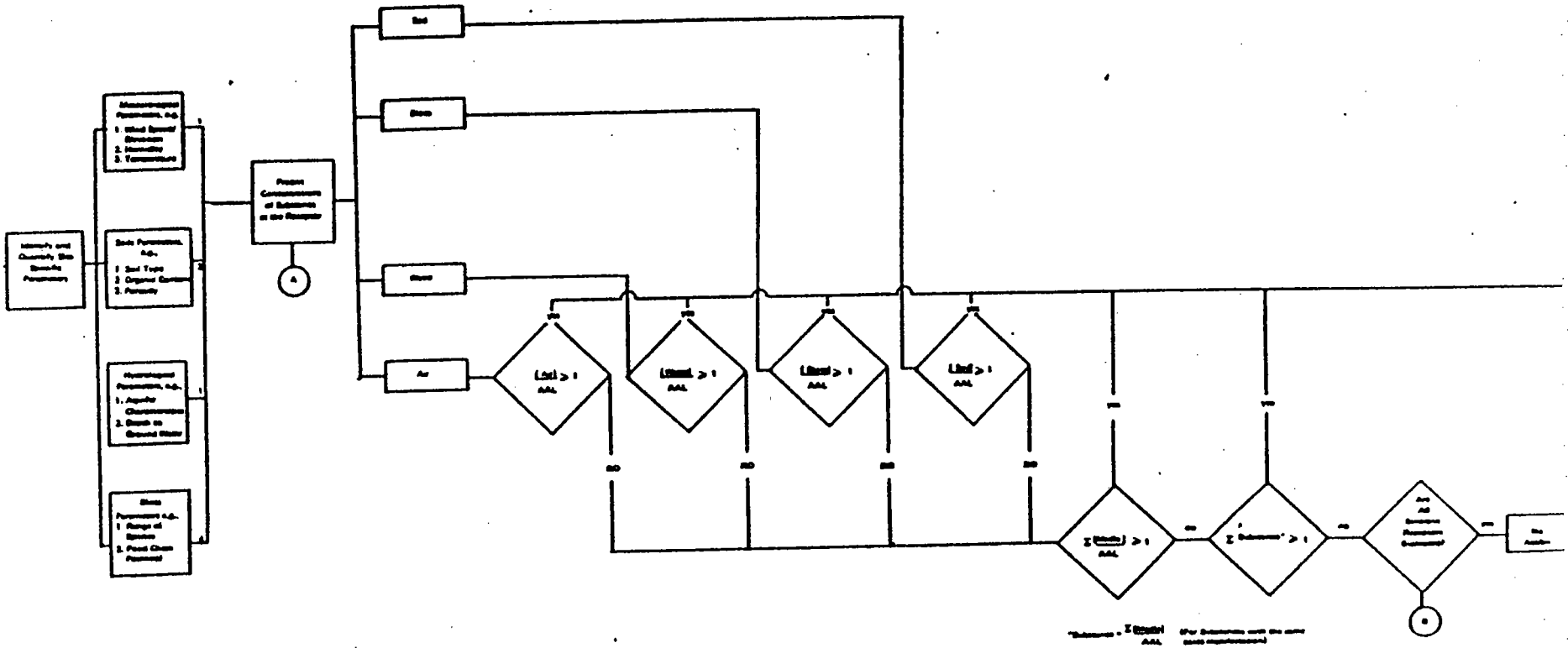


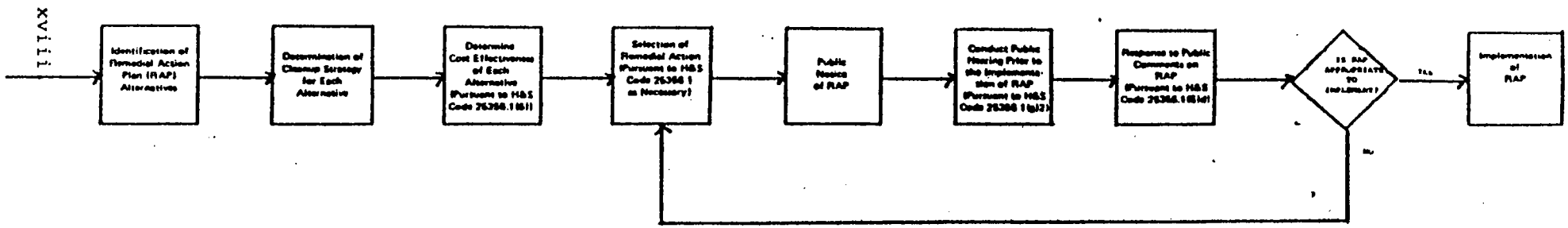
FIGURE 1. OVERVIEW OF THE DECISION TREE


Further Sampling and Analysis
No
All Chemical Contaminants Identified?
Yes





IV. ENVIRONMENTAL FATE AND RISK DETERMINATION



V. DEVELOPMENT OF MITIGATION STRATEGY AND SELECTION OF REMEDIAL ACTI

CHAPTER 9: REFERENCES

Baker, D.J. and R.A.F. Matheson. 1981. Dioxins in the Vicinity of Two Atlantic Region Wood Preserving Operations. Environmental Protection Service. EPS-5-AR-81-2. Nova Scotia, Canada.

Baughman, R.W. 1974. Tetrachlorodibenzo-p-dioxin in the environment. High resolution mass spectrometry of the picogram level. MA Thesis. Harvard University, Cambridge, MA.

Beatty, P.W., M.A. Holscher, and R.A. Neal. 1976. Toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin in larval and adult forms of Rana catesheriana. Bull. of Environ. Contam. and Toxicol. 16(5):578-581.

Beatty, P.W., W.K. Vaughn, and R.A. Neal. 1978. Effect of alteration of rat hepatic mixed-function oxidase (MFO) activity on the toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). Toxicol. Appl. Pharmacol. 45(2):513-519.

Bellin, J.S. and D.G. Barnes. 1986. Interim procedures for estimating risks associated with exposures to mixtures of chlorinated dibenzo-p-dioxins and dibenzofurans (CDDs and CDFs). Risk Assessment Forum, U.S. Environmental Protection Agency. EPA/625/3-87/012. Washington, DC.

Bowes, G.W., M.J. Mulvihill, B.R.T. Simoneit, A.L. Burlingame, and R.W. Risebrough. 1975. Identification of chlorinated dibenzofurans in American polychlorinated biphenyls. Nature 256:305-307.

Bowes, G.W., M.J. Mulvihill, B.R. Simoneit, A.L. Burlingame, and R.W. Risebrough. 1978. Isolation and identification of chlorinated dibenzofurans from polychlorinated biphenyls (PCB) and from Yusho rice oil containing PCB. Pp. 79-98 in F. Cattabeni, A. Carallaro, and G. Galli, eds., Dioxin: Toxicological and Chemical Aspects. Spectrum Publications, New York, NY.

Branson, D.R., I.T. Takahasi, W.M. Parker, and G.E. Blau. 1985. Bioconcentration kinetics of 2,3,7,8-tetrachlorodibenzo-p-dioxin in rainbow trout. Environ. Tox. and Chem. 4:779-788.

Bridle, T.R., B.K. Afghan, H.W. Campbell, R.J. Wilkinson, and J. Carron. 1984. The formation and fate of PCDDs and PCDFs during chlorophenol combustion. P. 84.11.2 in A. Sachdev, ed., Proceedings of the 77th Annual Meeting of the Air Pollution Control Association, San Francisco, CA.

Bruggeman, W.A., A. Opperhuizen, A. Wijbenga, and O. Hutzinger. 1984. Bioaccumulation of superlipophilic chemicals in fish. Toxicological and Environmental Chemistry 7:173-189.

Bumpus, J.A., M. Ting, D. Wright, and S.D. Aust. 1985. Oxidation of persistent environmental pollutants by a white rot fungus. Science 228:1434-1436.

Buser, H.R. 1976. Preparation of qualitative standard mixtures of polychlorinated dibenzo-p-dioxins and dibenzofurans by ultraviolet and gamma irradiation of the octachloro compounds. Journal of Chromatography 129:303-307.

Buser, H.R. 1979. Formation and identification of tetra- and pentachlorodibenzo-p-dioxins from photolysis of two isomeric hexachlorodibenzo-p-dioxins. Chemosphere 8:251-257.

Buser, H.R. and H.P. Bosshardt. 1976. Determination of polychlorinated dibenzofurans in commercial pentachlorophenols by combined gas chromatography/mass spectrometry. Journal of the AOAC 59(3):562-569.

Buser, H.R. and C. Rappe. 1978. Identification of substitution patterns in polychlorinated dibenzo-p-dioxins (PCDDs) by mass spectrometry. Chemosphere 7(2):199-211.

Buser, H.R., C. Rappe, and P.A. Bergqvist. 1985. Analysis of polychlorinated dibenzofurans, dioxins and related compounds in environmental samples. Env. Health Perspectives 60:293-302.

California Administrative Code. 1984. Title 22, Social Security. Division 4, Environmental Health, Article 11, Sections 66699 and 66700. Sacramento, CA.

California Air Resources Board (CARB) and California Department of Health Services (CDHS). 1986. Report to the Scientific Review Panel on Chlorinated Dioxins and Dibenzofurans. Prepared by the staffs of the CARB, Sacramento, California, and the CDHS. February 1986. Berkeley, CA.

California Central Valley Regional Water Quality Control Board. 1982. Inspection Report: Selma Pressure Treating Company, August 11. Sacramento, CA.

California Department of Health Services. May 1986. The California Site Mitigation Decision Tree Manual. Sacramento, CA.

California Department of Water Resources. 1982. The hydrologic-economic model of the San Joaquin Valley. Bulletin 214, State of California, December. Sacramento, CA.

Chinkin, L., B. Weir, and D. Latimer. March 1987. Inventory of Chlorophenol Use in the Forest Products Industry and Investigation of Related Emissions of Chlorinated Dibenzodioxins and Dibenzofurans. Report for the California Air Resources Board prepared by Systems Applications, Inc., San Rafael, CA.

Choudhry, G.G. and O. Hutzinger. 1982. Photochemical formation and degradation of polychlorinated dibenzofurans and dibenzo-p-dioxins. Residue Reviews 84:113-161.

Cocucci, S., F. DiGerolamo, A. Verderio, A. Cavallaro, G. Colli, A. Gorni, G. Invernizzi, and L. Luciani. 1979. Absorption and translocation of tetrachlorodibenzo-p-dioxin by plants from polluted soil. Experientia 35(4):482-484.

Commoner B., T. Webster, and K. Shapiro. 1986. Environmental levels and health effects of chlorinated dioxins and furans. Prepared for presentation at AAAS Annual Meeting, Philadelphia, PA, May 28, 1986. Center for the Biology of Natural Systems, Queens College, CUNY, Flushing, NY.

Corbet, R.L., D.C.G. Muir, and G.R.B. Webster. 1983. Fate of 1,3,6,8-T_4CDD in an outdoor aquatic system. Chemosphere 12(4/5):523-527.

Crosby, D.G. 1985. The degradation and disposal of chlorinated dioxins. Pp. 195-204 in M. Kamrin and P. Rogers, eds., Dioxins in the Environment. Hemisphere Publishing Company, New York, NY.

Crosby, D.G., K.I. Beynon, P.A. Greve, F. Korte, G.G. Still, and J.W. Vonk. 1981. Environmental chemistry of pentachlorophenol. Pure and Applied Chemistry 53:1051-1080.

Crosby, D.G., K.W. Moilanen, and A.S. Wong. 1973. Environmental generation and degradation of dibenzodioxins and dibenzofurans. Environmental Health Perspectives 5:259-266.

Crosby, D.G., A.S. Wong, J.R. Plimmer, and E.A. Woolson. 1971. Photodecomposition of chlorinated dibenzo-p-dioxins. Science 173:748-749.

Crummett, Warren. 1983. Status of analytical systems for the determination of PCDDs and PCDFs. Chemosphere 12(4/5): 429-446.

Cull, M.R. and A.J. Dobbs. 1984. Long term changes in polychlorodibenzo-p-dioxin concentrations in wood treated with technical pentachlorophenol. Chemosphere 13(a): 1091-1099.

Czuczwa, J.M., B.D. McVeety, and R.A. Hites. 1984. Polychlorinated dibenzo-p-dioxins and dibenzofurans in sediments from Siskiwit Lake, Isle Royale. Science 226: 568-569.

DaRos, B., W. Fitch, R. Merrill, and D. Wolbach. April 1981. Measured multimedia emissions from the wood preserving industry. Project summary prepared by the Acurex Corporation for the U.S. Environmental Protection Agency. Industrial Environmental Research Laboratory, Office of Research and Development, Cincinnati, OH.

DiDomenico, A., V. Silano, G. Viviano, and G. Zapponi. 1980a. Accidental release of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) at Seveso, Italy. IV. Vertical distribution of TCDD in soil. Ecotoxicology and Environmental Safety 4:327-338.

DiDomenico, A., V. Silano, G. Viviano, and G. Zapponi. 1980b. Accidental release of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) at Seveso, Italy. V. Environmental persistence of TCDD in soil. Ecotoxicology and Environmental Safety 4:339-345.

Dobbs, A.J. and C. Grant. 1979. Photolysis of highly chlorinated dibenzo-p-dioxins by sunlight. Nature 278:163-165.

Doull, J., C.D. Klaassen, and M.O. Amdur, eds. 1980. Casarett and Doull's Toxicology: The Basic Science of Poisons. MacMillan Publishing Co., New York, NY.

Enfield, C.G. 1985. Chemical transport facilitated by multi-phase flow systems. Wat. Sci. Tech. 17:1-12.

Esposito, M.P., T.O. Tiernan, and F.E. Dryden. 1980. Dioxins. EPA-600/2-80-197. Industrial Environmental Research Laboratory, Office of Research and Development, U.S. Environmental Protection Agency, Cincinnati, OH.

Facchetti, S., A. Balasso, C. Fichiner, G. Frare, A. Leoni, C. Mauri, and M. Vasconi. 1985. Assumption of 2,3,7,8-TCDD by Some Plant Species. Presented before the Division of Environmental Chemistry, American Chemical Society, Miami, FL.

Facchetti, S. and A. Leoni. 1980. Determination of 2,3,7,8-tetrachlorodibenzo-p-dioxin at the part per trillion level in biological and environmental samples. Pp. 215-243 in S. Facchetti, ed., Application of Mass Spectrometry to Trace Analysis. Joint Research Center, Ispra, Italy.

Firestone, D. 1978. The 2,3,7,8-tetrachlorodibenzo-p-dioxin problem: A review. Ecology Bulletin 27:39-52.

Freeman, R.A. and J.M. Schroy. 1985. Environmental mobility of dioxins. Pp. 420-439 in R.C. Bahner, and D.J. Hansen, eds., Aquatic Toxicology and Hazard Assessment: Eighth Symposium. A Symposium on Aquatic Toxicology and Hazard Assessment, ASTM STP 891, Philadelphia, PA.

Freeman, R.A. and J.M. Schroy. 1986. Modeling the transport of 2,3,7,8-TCDD and other low volatility chemicals in soils. Environmental Progress 5(1):28-33.

Goldstein, J.A. 1980. Structure activity relationships for the biochemical effects and the relationship to toxicity. Pp. 109-150 in R.D. Kimbrough, ed., Halogenated Biphenyls, Terphenyls, Naphthalenes, Dibenzodioxins, and Related Products. Elsevier/North-Holland, New York, NY.

Gray, A.P., S.P. Cepa, and J.S. Cantrell. 1975. Intervention of the Smiles rearrangement in synthesis of dibenzo-p-dioxins: 1,2,3,6,7,8- and 1,2,3,7,8,9-hexachlorodibenzo-p-dioxin (HCDD). Tetrahedron Letter 33:2873-2876.

Gray, A.P., S.P. Cepa, I.J. Solomon, and O. Aniline. 1976. Synthesis of specific polychlorinated dibenzo-p-dioxins. J. Org. Chem. 41:2435-2437.

Hardell, L., L. Domellof, M. Nygren, M. Hansson, and C. Rappe. 1985. Levels of polychlorinated dibenzodioxins and dibenzofurans in adipose tissue of patients with soft-tissue sarcoma or malignant lymphoma exposed to phenoxy acids and of unexposed controls. Paper presented at 189th National Meeting of the American Chemical Society, April 29 - May 3, 1985. Miami, FL.

Hawkes, C.L. and L. Norris. 1977. Chronic oral toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) to rainbow trout. Trans Am. Fish Soc. 106(6).

Heida, H. 1983. TCDD in bottom sediments and eel around a refuse dump near Amsterdam, Holland. Chemosphere 12(4/5):503-509.

Helder, T. 1980. Effects of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) on early life stages of the pike. The Science of the Total Environment 14:255-264.

Helder, T. 1981. Effects of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) on early stages of rainbow trout (Salmo gairdneri, Richardson). Toxicology 19:101-112.

Helder, T. 1982. The toxicity and toxic potential of fly ash from municipal incinerators assessed by means of a fish early life stage test. Chemosphere 11(10):965-972.

Hodgson, E. and F.E. Guthrie, eds. 1980. Introduction to Biochemical Toxicology. Elsevier/North-Holland, New York, NY.

Hoffman, R.E., P.A. Stehr-Green, K.B. Webb, R.G. Evans, A.P. Knutsen, W.F. Sachramm, J.L. Staake, B.B. Gibson, and K.K. Steinberg. 1986. Health effects of long-term exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin. JAMA 225(15):2031-2038.

Huetter, R. and M. Philippi. 1982. Studies on microbial metabolism of TCDD under laboratory conditions. Pp. 87-92 in O. Hutzinger, R.W. Frei, E. Merian, and F. Pocchiari, eds., Chlorinated Dioxins and Related Compounds: Impact on the Environment. Pergamon Press, New York, NY.

Huff, J.E., J.A. Moore, R. Saracci, and L. Tomatis. 1980. Long term hazards of polychlorinated dibenzodioxins and polychlorinated dibenzofurans. Environ. Health Perspect. 36:221-240.

Humppi, T., J. Knuutinen, and J. Paasivirta. 1984. Analysis of polychlorinated phenoxyphenols in technical chlorophenol formulations and in a sawmill environment. Chemosphere 13(11):1235-1241.

Hutzinger, O., M.J. Blumich, M.V.D. Berg, and K. Olie. 1985. Sources and fate of PCDDs and PCDFs: An overview. Chemosphere 14(6/7):581-600.

Hutzinger, O., S. Safe, B.R. Wentzell, and V. Zitko. 1973. Photochemical degradation of di- and octachlorodibenzofuran. Environmental Health Perspectives 5:267-273.

International Agency for Research on Cancer (IARC). 1982. Monographs on the evaluation of the carcinogenic risk of chemicals to humans. Pp. 238-243 in Chemical Industrial Priority and Industries Associated with Cancer in Humans. Suppl. 4. IARC, Lyon, France.

Isensee, A.R. 1978. Bioaccumulation of 2,3,7,8-tetrachlorodibenzo-para-dioxin. Ecol. Bull. 27:255-262. Stockholm, Sweden.

Isensee, A.R. and G.E. Jones. 1975. Distribution of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in aquatic model ecosystem. Environmental Science and Technology 9(7): 668-672.

Jackson, D.R., M.H. Roulier, H.M. Grotta, S.W. Rust, J.S. Warner, M.F. Arthur, and F.L. DeRoos. 1985. Leaching potential of 2,3,7,8-TCDD in contaminated soils. Pp. 153-168 in Land Disposal of Hazardous Waste. Proceedings of the Eleventh Annual Research Symposium. No. EPA/600/9-85/013, U.S. Environmental Protection Agency. Washington, DC.

Jaegar, Rudolph J. 1984. Summary to toxicity literature and evaluation of health risk due to chlorinated contaminants in drinking water. Prepared for Southern California Edison Co., Rosemead, CA.

Kaczmar, S.W., M.J. Zabik, and F.M. D'Itri. 1985. Part per trillion residues of 2,3,7,8-tetrachlorodibenzo-p-dioxin in Michigan fish. Pp. 103-110 in L.H. Keith, C. Rappe, and G. Choudhary, eds., Chlorinated Dioxins and Dibenzofurans in the Total Environment II. Butterworth Publishers, Boston, MA.

Karickhoff, S.W. and K.R. Morris. 1985. Sorption dynamics of hydrophobic pollutants in sediment suspensions. Environmental Toxicology and Chemistry 4:469-479.

Kenaga, E.E. and L.A. Norris. 1983. Environmental toxicity of TCDD. Pp. 277-299 in R.E. Tucker, A.L. Young, and A.P. Gray, eds., Human and Environmental Risks of Chlorinated Dioxins and Related Compounds. Plenum Press, New York, NY.

Kende, A.S., J.J. Wade, D. Ridge, and A. Pohland. 1974. Synthesis and fourier transform carbon-13 nuclear magnetic resonance spectroscopy of new toxic polyhalodibenzo-p-dioxins. Journal Organic Chemistry 39:931-937.

Kerkvliet, N.I., J.A. Brauner, and J.P. Matlock. 1985. Humoral immunotoxicity of polychlorinated diphenyl ethers, phenoxy-phenols, dioxins and furans present as contaminants of technical grade pentachlorophenol. Toxicology 36:307-324.

Kimbrough, R.D., H. Falk, P. Strehr, and G. Fries. 1984. Health implications of 2,3,7,8-tetrachlorodibenzodioxin (TCDD) contamination of residential soil. Journal of Toxicology and Env. Health 14:47-93.

Kitunen, V.H., R.J. Valo, and M.S. Salkinoja-Salonen. 1987. Contamination of soil around wood-preserving facilities by polychlorinated aromatic compounds. Environ. Sci. Technol. 21:96-101.

Kleeman, J.M., J.R. Olson, S.M. Chen, and R.E. Peterson. 1986a. Metabolism and disposition of 2,3,7,8-tetrachlorodibenzo-p-dioxin in rainbow trout. Toxicology and Applied Pharmacology 83:391-401.

Kleeman, J.M., J.R. Olson, S.M. Chen, and R.E. Peterson. 1986b. 2,3,7,8-tetrachlorodibenzo-p-dioxin metabolism and disposition in yellow perch. Toxicology and Applied Pharmacology 83:402-411.

Kleopfer, Robert D. 1985. 2,3,7,8 contamination in Missouri. Chemosphere 14(6/7):739-744.

Kociba, R.J. 1984. Summary and critique of rodent carcinogenicity studies of chlorinated dibenzo-p-dioxins. Pp. 77-98 in W.K. Lowrance, ed., Public Health Risks of the Dioxins. William Kaufman, Los Altos, CA.

Kuehl, D.W., P. Cook, and A.R. Batterman. 1985a. Uptake and depuration studies of PCDDs and PCDFs in freshwater fish. Draft report, Environmental Protection Agency, Duluth, MN.

Kuehl, D.W., P.M. Cook, A.R. Batterman, and B.C. Butterworth. 1986a. Isomer dependent availability of polychlorinated dibenzo-p-dioxins and dibenzofurans from municipal incinerator fly ash to carp. (Submitted for publication to Chemosphere.)

Kuehl, D.W., P.M. Cook, A.R. Batterman, and D.B. Lothenbach. 1985b. Bioavailability of 2,3,7,8-tetrachlorodibenzo-p-dioxin from municipal incinerator fly ash to freshwater fish. Chemosphere 14(5):427-437.

Kuehl, D.W., P.M. Cook, A.R. Batterman, D. Lothenbach, and B.C. Butterworth. 1986b. Bioavailability of polychlorinated dibenzo-p-dioxins and dibenzofurans from contaminated Wisconsin River sediment to carp. (Submitted for publication to Chemosphere.)

Kuroki, H., Y. Matsuda, S. Yoshihara, and H. Yoshimura. 1980. Accumulation of polychlorinated dibenzofurans in the livers of monkeys and rats. Fd. Cosmet. Toxicol. 18:387-392.

Lamparski, L.L., R.J. Stehl, and R.L. Johnson. 1980. Photolysis of pentachlorophenol-treated wood. Chlorinated dibenzo-p-dioxin formation. Environ. Sci. Technol., 14:196-200.

Lao, R.C., R.S. Thomas, C. Chiu, and K. Li. 1985. Analysis of PCDD-PCDF in environmental samples. Pp. 65-78 in G. Choudhary, L.H. Keith, and C. Rappe, eds., Chlorinated Dioxins and Dibenzofurans in the Total Environment II. Butterworth Publishers, Boston, MA.

Levin, J.O., C. Rappe, and C.A. Nilsson. 1976. Use of chlorophenols as fungicides in sawmills. Scand. J. World Environ. Health 2:71-81.

Lucier, G.W., R.C. Rumbaugh, Z. McCoy, R. Hass, D. Harvan, and P. Albro. 1986. Ingestion of soil contaminated with 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) alters hepatic enzyme activities in rats. Fundamental and Applied Toxicology 6:364-371.

Marcheterre, L., G.R.B. Webster, D.C.G. Muir, and N.P. Grift. 1985. Fate of ^{14}C-octachlorodibenzo-p-dioxin in artificial outdoor ponds. Chemosphere 14:835-838.

Masuda, Y. and H. Yoshimura. 1984. Polychlorinated biphenyls and dibenzofurans in patients with Yusho and their toxicological significance: A review. American Journal of Industrial Medicine 5:31-44.

Matsumura, Fumio. 1977. Absorption, accumulation, and elimination of pesticides by aquatic organisms. Environmental Science Research 10:77-105.

Matsumura F. and H.J. Benezet. 1973. Studies on the Bioaccumulation and microbial degradation of 2,3,7,8-tetrachlorodibenzo-p-dioxin. Environmental Health Perspectives 5:253-258.

May, G. 1973. Chloracne from the accidental production of tetrachlorodibenzodioxin. British J. Industrial Medicine. 30:276-283.

Mazer, T. and F.D. Hileman. 1982. The ultraviolet photolysis of tetrachlorodibenzofurans. Chemosphere 11(7):651-661.

McConnell, E.E. 1980. Acute and chronic toxicity, carcinogenesis, reproduction, teratogenesis, and mutagenesis in animals. Pp. 109-150 in R.D. Kimbrough, ed., Halogenated Biphenyls, Terphenyls, Naphthalenes, Dibenzodioxins and Related Products. Elsevier/North-Holland, Biomedical Press, New York, NY.

McConnell, E.E., G.W. Lucier, R.C. Rumbaugh, P.W. Albro, D.J. Harvan, J.R. Hass, and M.W. Harris. 1984. Dioxin in soil: bioavailability after ingestion by rats and guinea pigs. Science 223:1077-1079.

McKinney, J. and E. McConnell. 1982. Structural specificity and the dioxin receptor. Pp. 367-381 in O. Hutzinger, R.W. Frei, E. Merian, and F. Pocchiari, eds., Chlorinated Dioxins and Related Compounds: Impact on the Environment. Pergamon Press, New York, NY.

McNulty, W.P. 1985. Toxicity and fetotoxicity of TCDD, TCDF and PCB isomers in rhesus macaques (Macaca mulatta). Env. Health Persp. 60:77-85.

Mehrle, P.M., D.R. Buckler, E.E. Little, L.M. Smith, J.D. Petty, P.H. Peterman, D.L. Stalling, G.M. DeGraeve, J.J. Coyle, and W.J. Adams. 1988. Toxicity and bioconcentration of 2,3,7,8-tetrachlorodibenzodioxin and 2,3,7,8-tetrachlorodibenzofuran in rainbow trout. Environmental Toxicology and Chemistry. 7:47-62.

Michigan Department of Public Health. 1986. Background Document Concerning 1986 Fish Consumption Advisory for Dioxin Contaminated Fish. February. Lansing, MI.

Miles, W.F., N.P. Gurprasad, and G.P. Malis. 1985a. Isomer specific determination of hexachlorodibenzo-p-dioxins by oxygen negative chemical ionization, mass spectrometry, gas chromatography, and high-pressure liquid chromatography. Analytical Chemistry 57:1133-1138.

Miles, W.F., J. Singh, N.P. Gurprasad, and G.P. Malis. 1985b. Isomer specific determination of hexachlorodioxins in technical pentachlorophenol (PCP) and its sodium salt. Chemosphere 14(6/7):807-810.

Miller, R.A., L.A. Norris, and C.L. Hawkes. 1973. Toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in aquatic organisms. Env. Health Perspectives 5:177.

Miller, R.A., L.A. Norris, and B.R. Loper. 1979. The response of coho salmon and guppies to 2,3,7,8-tetrachlorodizeno-p-dioxin (TCDD) in water. Transaction of the American Fisheries 108:401-407.

Morgan III, Joe. Letter dated November 17, 1986 from J. Morgan III of J.H. Baxter and Company to Frank Palmer of the State Water Resources Control Board. Sacramento, CA.

Muir, D.G.G., B.E. Townsend, and G.R.B. Webster. 1984. Bioavailability of ^{14}C-1,3,6,8-tetrachlorodibenzo-p-dioxin and ^{14}C-octachlorodibenzo-p-dioxin to aquatic insects in sediment and water. Pp. 89-102 in Chlorinated Dioxins and Dibenzofurans in the Total Environment II. Butterworth Publishers, Boston, MA.

Muir, D.C.G., A.L. Yarechewski, R.L. Corbet, G.R.B. Webster and A.E. Smith. 1985a. Laboratory and field studies on the fate of 1,3,6,8-tetrachlorodibenzo-dioxin in soil and sediments. J. Agric. Food Chem. 33:518-523.

Muir, D.C.G., A.L. Yarechewski, A. Knoll, and G.R.B. Webster. 1986. Bioconcentration and disposition of 1,3,6,8-tetrachlorodibenzo-p-dioxin and octachlorodibenzo-p-dioxin by rainbow trout and fathead minnows. Environmental Toxicology and Chemistry 5:261-272.

Muir, D.C.G., A.L. Yarechewski, and G.R.B. Webster. 1985b. Bioconcentration of four chlorinated dioxins by rainbow trout and fathead minnows. Pp. 440-454 in R.C. Bahner and D.J. Hansen, eds. Aquatic Toxicology and Hazard Assessment: Eighth Symposium, ASTM, STP 891. American Society for Testing and Materials. Philadelphia, PA.

National Academy of Sciences. 1977. Drinking Water and Health. Safe Drinking Water Committee, Advisory Center on Toxicology, Assembly of Life Sciences, National Research Council, National Academy of Sciences, Washington, DC.

National Institute for Occupational Safety and Health (NIOSH). 1983. Registry of Toxic Effects of Chemical Substances. Volumes 1-3. Cincinnati, OH.

National Research Council of Canada (NRCC). 1981. Polychlorinated Dibenzo-p-dioxins: Criteria for their Effects on Humans and the Environment. NRCC No. 18574. Ottawa, Canada.

National Research Council of Canada (NRCC). 1984. Polychlorinated Dibenzofurans: Criteria for their Effects on Humans and the Environment. NRCC No. 22846. Ottawa, Canada.

National Toxicology Program. 1982a. Carcinogenesis Bioassay of (CAS No. 1746-01-6) 2,3,7,8-tetrachlorodibenzo-p-dioxin in Osborne-Mendle Rats and B6C3F1 Mice (Gavage Study). NIH Publ. No. 82-1765. National Toxicology Program, Research Triangle Park, NC.

National Toxicology Program. 1982b. Carcinogenesis Bioassay of 2,3,7,8-Tetrachlorodibenzo-p-dioxin (CAS No. 1746-01-6) in Swiss-Webster Mice (Dermal Study). NIH Publ. No. 82-1757. National Toxicology Program, Research Triangle Park, NC.

Nestrick, T.J., L.L. Lamparski, and D.I. Townsend. 1980. Identification of tetrachlorodibenzo-p-dioxin isomers at the 1-ng level by photolytic degradation and pattern recognition techniques. Analytical Chemistry 52:1865-1874.

New York State Legislature. 1985. Amendments to Surface Water Quality Standards (Revised 6NYCRR 701-702, effective August 2, 1985.) Albany, NY.

Niimi, A.J. and B.G. Oliver. 1986. Biological half-lives of chlorinated dibenzo-p-dioxins and dibenzofurans in rainbow trout (Salmo gairdneri). Environmental Toxicology and Chemistry 5:49-53.

Nilsson, C.A., K. Anderson, C. Rappe, and S.O. Westermark. 1974. Chromatographic evidence for the formation of chlorodioxin from chloro-2-phenoxyphenols. J. Chrom. 96:137-147.

Nilsson, C.A., A. Norstrom, K. Anderson, and C. Rappe. 1978. Impurities in commercial products related to pentachlorophenol. Env. Sci. Res. 12:313-24.

Nkedi-Kizza, P., P.S.C. Rao, and A.G. Hornsby. 1985. Influence of organic cosolvents on sorption of hydrophobic organic chemicals by soils. Environ. Sci. Technol. 19:975-979.

Norris, L.A. and R.A. Miller. 1974. The toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in guppies. Bulletin of Environmental Contamination and Toxicology 12(1):76.

Norstrom, A., K. Andersson, and C. Rappe. 1976. Formation of chlorodibenzofurans by irradiation of chlorinated diphenyl ethers. Chemosphere 5:21-24.

Norstrom, A., K. Andersson, and C. Rappe. 1977. Studies on the formation of chlorodibenzofurans by irradiation or pyrolysis of chlorinated diphenyl ethers. Chemosphere 6:241-248.

Nygren, M., M. Hansson, C. Rappe, L. Dommellof, and L. Hardell. 1985. Analysis of polychlorinated dibenzo-p-dioxins and dibenzofurans in adipose tissue from soft-tissue sarcoma patients and controls. Paper presented at 189th National Meeting of the American Chemical Society, April 29 - May 3, 1985. Miami. FL.

O'Keefe, P., D. Hilker, C. Meyer, K. Aldous, L. Shane, R. Donnely, and R. Smith. 1984. Tetrachlorodibenzo-p-dioxins and tetrachlorodibenzofurans in Atlantic coast striped bass and in selected Hudson River fish, waterfowl, and sediments. Chemosphere 13(8):849-860.

Ontario Ministry of the Environment. 1986. Guide to Eating Ontario Sport Fish. Ministry of the Environment, St. Clair, Toronto, Canada.

Parkinson, A., and S. Safe. 1981. Aryl hydrocarbon hydroxylase induction and its relationship to the toxicity of halogenated aryl hydrocarbons. Toxicology and Chemical Reviews 4:1-26.

Pereira, W.E., C.E. Rostad, and M.E. Stark. 1985. Geochemical investigations of polychlorinated dibenzo-p-dioxins in the subsurface environment at an abandoned wood treatment facility. Environmental Toxicology and Chemistry 4:629-639.

Plimmer, J.R. 1978. Photolysis of TCDD and trifluralin on silica and soil. Bull. Environ. Contam. Toxicol. 20:87-92.

Podoll, R. Thomas, H.M. Jaber, and T. Mill. 1986. Tetrachlorodibenzodioxin: rates of volatilization and photolysis in the environment. Environ. Sci. Technol. 20(5):490-552.

Pohland, A.E. and G.C. Yang. 1972. Preparation and characterization of chlorinated dibenzo-p-dioxins. Agric. Food Chemistry 20(6):1093-1099.

Poiger, H. and C. Schlatter. 1983. Animal toxicology of chlorinated dibenzo-p-dioxins. Chemosphere 12(4/5):453-462.

Poiger, H. and C. Schlatter. 1986. Pharmacokinetics of 2,3,7,8-TCDD in man. Chemosphere 15(9-12):1489-1498.

Quensen III, J.F., and F. Matsumura. 1983. Oxidative degradation of 2,3,7,8-tetrachlorodibenzo-p-dioxin by microorganisms. Environmental Toxicology and Chemistry 2:261-268.

Rappe, Christopher. 1984. Analysis of polychlorinated dioxins and furans. Environ. Sci. Tech. 18(3).

Rappe, C., H.R. Buser, and H.P. Bosshardt. 1979. Env. Sci. Tech. 18(3):78A-90A.

Rappe, C., S. Marklund, M. Nygren, A. Gara. 1983a. Parameters for identification and confirmation in trace analyses of polychlorinated dibenzo-p-dioxins and dibenzofurans. Pp. 259-272 in G. Choudhary, L.H. Keith, and C. Rappe, eds., Chlorinated Dioxins and Dibenzofurans in the Total Environment. Butterworth Publishers, Boston, MA.

Rappe, C., M. Nygren, and C. Gustafsson. 1983b. Human exposure to polychlorinated dibenzo-p-dioxins and dibenzofurans. Pp. 355-365 in G. Choudhary, L.H. Keith, and C. Rappe, eds., Chlorinated Dioxins and Dibenzofurans in the Total Environment. Butterworth Publishers, Boston, MA.

Reggiani, G. 1980. Localized contamination with TCDD - Seveso, Missouri and other areas. Pp. 303-372 in R. D. Kimbrough, ed., Halogenated Biphenyls, Terphenyls, Naphthalenes, Dibenzodioxins, and Related Products. Elsevier/North-Holland, Biomedical Press, New York, NY.

Roberts, E.A., N.H. Shear, and A.B. Okey. 1985. The Ah receptor and dioxin toxicity: From rodent to human tissues. Chemosphere 14(6/7):661-674.

Rose, M.E. and R.A.W. Johnstone. 1982. Mass Spectrometry for Chemists and Biochemists. Cambridge University Press, Cambridge, England.

Sarna, L.P., P.E. Hodge, and G.R.B. Webster. 1984. Octanol-water partition coefficients of chlorinated dioxins and dibenzofurans by reversed phase HPLC using several C_{18} columns. Chemosphere 13(9):975-983.

Schecter, A., J.J. Ryan, R. Lizotte, W.F. Sun, L. Miller, G. Gitlitz, and M. Bogdasarian. 1985a. Chlorinated dibenzodioxins and dibenzofurans in human adipose tissue from exposed and control New York state patients. Chemosphere 14(6/7):933-937.

Schecter, A., T.O. Tiernan, M.L. Taylor, G.F. Van Ness, J.H. Garrett, D.J. Wagel, G. Gitlitz, and M. Bagdasarian. 1985b. Biological markers after exposure to polychlorinated dibenzo-p-dioxins, dibenzofurans, biphenyls, and biphenylenes. Part 1: Findings using fat biopsies to estimate exposure. Pp. 215-245 in L.H. Keith, C. Rappe, and G. Choudhary, eds., Chlorinated Dioxins and Dibenzofurans in the Total Environment II. Butterworth Publishers, Boston, MA.

Smith, L.M., and J.L. Johnson. 1983. Evaluation of interferences from seven series of polychlorinated aromatic compounds in an analytical method for polychlorinated dibenzofurans and dibenzo-p-dioxins in environmental samples. Pp. 321-332 in G. Choudhary, L.H. Keith, and C. Rappe, eds., Chlorinated Dioxins and Dibenzofurans in the Total Environment. Butterworth Publishers, Boston.

Southern California Edison. 1983. Ground Water Quality Conditions at the SCE Visalia Pole Yard During the Year. Rosemead, CA.

1984. Annual Report of the SCE Visalia Pole Yard and Related Issues. Rosemead, CA.

1985a. First Quarterly Report on the SCE Visalia Pole Yard. Rosemead, CA.

1985b. Third Quarterly Report on the SCE Visalia Pole Yard. Rosemead, CA

1985c. Fourth Quarterly Report on the SCE Visalia Pole Yard. Rosemead, CA.

1985d. Annual Report of the SCE Visalia Pole Yard and Related Issues. Rosemead, CA.

1986. Report on Sampling and Analytical Results of Sites Suspected of Using City of Visalia Sewage Sludge. Rosemead, CA.

Thibodeaux, L.J. 1983. Offsite transport of 2,3,7,8-tetrachlorodibenzo-p-dioxin from a production disposal facility. Pp. 75-86 in G. Choudhary, L.H. Keith, and C. Rappe, eds., Chlorinated Dioxins and Dibenzofurans in the Total Environment. Butterworth Publishers, Boston, MA.

Tiernan, Thomas O. 1983. Analytical chemistry of polychlorinated dibenzo-p-dioxins and dibenzofurans: A review of the current status. Pp. 211-237 in G. Choudhary, L.H. Keith, and C. Rappe, eds., Chlorinated Dioxins and Dibenzofurans in the Total Environment. Butterworth Publishers, Boston, MA.

Tiernan, T.O., M.L. Taylor, J.H. Garrett, G.F. Van Ness, J.G. Solch, D.A. Deis, and D.J. Wagel. 1983. Chlorobenzodioxins, chlorobenzofurans, and related compounds in the effluents from combustion processes. Chemosphere 12(4/5):595-606.

Tsushimoto, G., F. Matsumura and R. Sago. 1982. Fate of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in an outdoor pond and in model aquatic ecosystems. Environmental Toxicology and Chemistry 1:61-68.

Umbreit, T.H., E.J. Hesse, M.A. Gallo. 1986. Bioavailability of dioxin in soil from a 2,4,5-T manufacturing site. Science 232:497-499.

U. S. Congress. 1983. Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA). Federal Register 48:FR 48434. Congress, Washington, DC.

U. S. Environmental Protection Agency. 1978. Report on the Ad Hoc Study Group on Pentachlorophenol Contaminants. EPA/SAB/78/001. Environmental Health Advisory Committee, Science Advisory Board, Washington, DC.

1979. Water-Related Environmental Fate of 129 Priority Pollutants. EPA-440/4-79-029a. Office of Water Planning and Standards, Washington, DC.

1981. Timber Products Processing Point Source Category. Federal Register, 40 CFR, 429; Volume 46, No. 16:8260-8294. Monday, January 26, 1981.

1983. Hazardous Waste Management System; Prohibition of Disposal of TCDD, Proposed Rule. Federal Register, 40 CFR, 261, 264, 265, and 775; Volume 48, No. 65:14514-14529. Monday, April 4, 1983.

1984a. Ambient Water Quality Criteria for 2,3,7,8-Tetrachloro- dibenzo-p-dioxin. EPA 440/5-84-007. Office of Water Regulations and Standards, Washington, DC.

1984b. National Dioxin Study. Tier 4 Combustion Sources, Initial Literature Review and Testing Options. U.S. EPA, OAQPS. October. Washington, DC.

1985a. Hazardous Waste Management System; Dioxin Containing Wastes; Rule. Federal Register, 40 CFR, 261, 264, 265, 270, 775; Vol. 50, No. 9:1978-2006. January 14, 1985. Washington, DC.

1985b. Health Assessment Document for Polychlorinated Dibenzo-p-dioxins, EPA/600/8-84-014F, Office of Health and Environmental Assessment, Cincinnati, OH.

1985c. National Primary Drinking Water Regulations; Synthetic Organic Chemicals, Inorganic Chemicals and Microorganisms, Proposed Rule. Federal Register, 40 CFR, Part 141, Vol. 50, No. 219:46819-46933. Wednesday, November 13, 1985. Washington, DC.

1986a. Alternate Concentration Limits for Dioxin Ground Water Contamination at Wood Preserving Sites. Memo from Cate Jenkins, Ph.D., Waste Identification Branch. Office of Solid Waste Emergency Response, U. S. Environmental Protection Agency, Washington, DC.

1986b. Hazardous Waste Management System; Land Disposal Restrictions, Final Rule. Federal Register 40 CFR Part 260, Volume 51, No. 216:40572-40654. Friday, November 7, 1986. Washington, DC.

1986c. Quality Assurance Report--Selma Site. August 8, 1986. Prepared by Lockheed Engineering and Management Services Company, Inc. Las Vegas, NV.

1986d. Sampling Plan: U. S. Environmental Protection Agency Enforcement Support Services for the Selma Pressure Treating Site. Work Assignment No. 24-9L440. March 19, 1986. Prepared by Camp Dresser and McKee Inc. Selma, CA.

1986e. Letter from Kathleen G. Shimmin, Chief Field Operations Branch, Toxics and Waste Management Division to John Youngerman, Division of Water Quality, State Water Resources Control Board, regarding levels of dioxin in fish tissues and sediment in the Santa Ana River. Region 9, San Francisco, CA.

1986f. Personal communication, Steve Simanonok, U. S. Environmental Protection Agency, Region 9, San Francisco, CA.

1987. Pentachlorophenol: Amendment of Notice of Intent to Cancel Registrations. Federal Register, 40 CFR; Vol. 52, No. 1:140-148.

U. S. Fish and Wildlife Service. 1983. Impacts of Contaminants on Early Life Stages of Striped Bass. Progress Report 1980-1983. Columbia National Fisheries Research Laboratory, Columbia, MO.

U. S. Food and Drug Administration. 1983. Statement by S.A. Miller, Director, Bureau of Foods, FDA, before the Subcommittee on Natural Resources, Agriculture Research and Environment, U. S. House of Representatives, June 30, 1983. Washington, DC.

Veith, G.D., K.J. Macek, S.R. Petrocelli, and J. Carrol. 1980. An evaluation using partition coefficients and water solubility to estimate bioconcentration factors for organic chemicals in fish. Pp. 116-129 in J.G. Eaton, P.R. Parrish, and A.C. Hendricks, eds., Aquatic Toxicology, STP 707. American Society for Testing and Materials. Philadelphia, PA.

Veith, G.D. and P. Kosian. 1983. Estimating bioconcentration potential from octanol/water partition coefficients. Pp. 269-282 in D. Mackey, S. Paterson, S.J. Eisenreich, and M.S. Simmons, eds., Physical Behavior of PCBs in the Great Lakes. Ann Arbor Science, Ann Arbor, MI.

Vickers, A.E., T.C. Sloop, and G.W. Lucier. 1985. Mechanism of action of toxic halogenated aromatics. Env. Health Perspect. 59:121-128.

Wipf, H.K., E. Homberger, N. Neuner, U.B. Renalder, W. Vetter, and J.P. Vuilleumier. 1982. TCDD levels in soil and plant samples from the Seveso area. Pp. 115-126 in O. Hutzinger, R.W. Frei, E. Merian, and F. Pocchiari, eds., Chlorinated Dioxins and Related Compounds: Impact on the Environment. Pergamon Press, New York.

Wolfe, W.H., G.D. Lathrop, R.A. Albanese, and P.M. Moynahan. 1985. An epidemiologic investigation of health effects in Air Force personnel following exposure to herbicides and associated dioxins. Chemosphere 14(6/7):707-716.

Wong, T.S. 1984. Dioxin formation and destruction in combustion processes. P. 84.12.7 in the Proceedings of the 77th Annual Meeting of the Air Pollution Control Association, San Francisco, CA.

World Health Organization. 1985. IPCS environmental health criteria, polychlorinated dibenzo-para-dioxins and dibenzofurans. Draft report, United Nations Environment Programme, International Labour Organization. New York, NY.

Yamagishi, T., T. Miyazaki, and K. Akiyama. 1981. Polychlorinated dibenzo-p-dioxins and dibenzofurans in commercial diphenyl ether herbicides, and in fresh water fish collected from the application area. Chemosphere 10:1137-1144.

Yockim, R.S., A.R. Isensee, and G.E. Jones. 1978. Distribution and toxicity of TCDD and 2,4,5-T in an aquatic model ecosystem. Chemosphere 7(3):215-220.

Young, A.L. 1981. Long term studies on the persistence and movement of TCDD in a natural ecosystem. Env. Sci. Res. 26:173-190.

Zambrano, J. 1987. Personal Communication, J. Zambrano of the New York State Department of Environmental Conservation, with Judith Heath, State Water Resources Control Board. Sacramento, CA.

Zitko, V. and P.M.K. Choi. 1973. Oral toxicity of chlorinated dibenzofurans to juvenile Atlantic salmon. Bulletin of Environmental Contamination and Toxicology 10(2):120.

Zitko, V., D.J. Wildish, O. Hutzinger, and P.M.K. Choi. 1973. Acute and chronic oral toxicity of chlorinated dibenzofurans to salmonid fishes. Env. Health Perspectives 5:187.

APPENDIX A

PHYSICAL AND CHEMICAL CHARACTERISTICS OF CDDs AND CDFs

APPENDIX A

PHYSICAL AND CHEMICAL PROPERTIES of CDDs AND CDFs

The chemical structure and nomenclature of CDDs and CDFs are discussed in the Introduction (Chapter 1). The following discussion is referenced to material presented in Table A.1 of this Appendix.

COMPOUND AND MOLECULAR FORMULA

Of the 75 CDD congeners and 135 CDF congeners, only 28 CDD and 3 CDF congeners have been found to have experimentally derived data on physical and chemical properties. The lack of adequate identification and characterization of CDD and CDF compounds stems largely from analytical problems (Monitoring Chapter).

CAS NUMBER

The Chemical Abstract Service (CAS) numbers are from the publication "Registry of Toxic Effects of Chemical Substances" (NIOSH, 1983). Each CAS number in Table A.1 identifies a specific compound. This unique number, with the aid of a computer, gives the reader rapid access to toxicity and chemical information necessary for the preparation of safety measures or hazard evaluations for these substances.

MOLECULAR WEIGHT

As the number of chlorine atoms in a compound increases, the molecular weight of that compound increases correspondingly. MonoCDD, with only one chlorine atom, weighs 218.64 grams/-mole, whereas octaCDD, with eight chlorine atoms, weighs 459.72 grams/mole.

PHYSICAL STATE

At standard temperature and pressure CDDs are in solid, colorless form, usually appearing in the shape of crystals or needles. The physical states of CDFs have not been adequately described in the literature.

MELTING POINT

The CDDs and CDFs are considered to be very stable and resistant towards heat. The less chlorinated CDD compounds begin to melt at 80° to 90°C, whereas the more highly chlorinated compounds generally melt above 200°C.

VAPOR PRESSURE

The literature gives a wide range of vapor pressure values. Values given for 2,3,7,8-tetraCDD are: 1.5×10^{-7} mm Hg (25°C)

TABLE A.1

PHYSICAL AND CHEMICAL CHARACTERISTICS OF CDDs AND CDFs

Compound/ Molecular Formula	CAS Number	Molecular Weight (grams/ mole)	Physical State (at Standard Temperature(^{o}C) and Pressure)	Melting Point	Vapor Pressure (mmHg)	Log K_{ow} (Octanol/ Water Partition Coefficient[a])	Solubility in Water (ug/l)	Reference
CDDs								
monoCDD $C_{12}H_7ClO_2$	39227-53-7	218.64	Colorless Crystals	80-90	N/A[d]	N/A	N/A	NIOSH, 1983
diCDD $C_{12}H_6Cl_2O_2$	39227-54-8	253.08	Colorless Solid	88-89	N/A	5.6	N/A	Sarna, et al., 1984; NIOSH, 1983; Pohland and Yang, 1972
1,3-diCDD $C_{12}H_6Cl_2O_2$	50585-39-2	253.08	Colorless Solid	113.5 - 114.5	N/A	N/A	N/A	Kende et al., 1974; NIOSH, 1983
1,6-diCDD $C_{12}H_6Cl_2O_2$	38178-38-0	253.08	Colorless Needles	N/A	N/A	N/A	N/A	NIOSH, 1983
2,3-diCDD $C_{12}H_6Cl_2O_2$	29446-15-9	253.08	Colorless Solid	163 - 164	N/A	N/A	N/A	Pohland and Yang, 1972; NIOSH, 1983
2,7-diCDD $C_{12}H_6Cl_2O_2$	33857-26-0	253.08	Colorless Crystals	209 - 210	7.0×10^{-6}	6.5	N/A	Sarna et al., 1984; NIOSH, 1983; Pohland and Yang, 1972
2,8-diCDD $C_{12}H_6Cl_2O_2$	38964-22-6	253.08	Colorless Solid	143 - 150	6.8×10^{-6}	N/A	N/A	Pohland and Yang, 1972; NIOSH, 1983
1,2,4-tri-CDD $C_{12}H_5Cl_3O_2$	39227-58-2	287.52	Colorless Solid	128-129	N/A	7.6	N/A	Pohland and Yang, 1972; NIOSH, 1983; Sarna et al., 1984
2,3,7-tri-CDD $C_{12}H_5Cl_3O_2$	33857-28-2	287.52	N/A	157-158	3.6×10^{-6}	N/A	N/A	Gray et al., 1976; Kende et al., 1974; NIOSH, 1983 U.S. EPA, 1978

TABLE A.1 (continued)

PHYSICAL AND CHEMICAL CHARACTERISTICS OF CDDs AND CDFs

Compound/ Molecular Formula	CAS Number	Molecular Weight (grams/ mole)	Physical State (at Standard Temperature(^{o}C) and Pressure)	Melting Point	Vapor Pressure (mmHg)	Log K_{ow} (Octanol/ Water Partition Coefficient[a])	Solubility in Water (ug/l)	Reference
Page 2								
CDDs								
1,2,3,4-tetraCDD $C_{12}H_4Cl_4O_2$	30746-58-8	321.96	Colorless Needles	188-190	N/A	8.8	N/A	Sarna et al., 1984; Pohland and Yang 1972; NIOSH, 1983
1,2,3,8-tetraCDD $C_{12}H_4Cl_4O_2$	53555-02-5	321.96	N/A	N/A	N/A	N/A	N/A	NIOSH, 1983
1,3,6,8-tetraCDD[b] $C_{12}H_4Cl_4O_2$	33423-92-6	321.96	Colorless Needles	219-219.5	N/A	9.0-9.26	0.353	Sarna et al., 1984; Pohland and Yang, 1972; NIOSH, 1983; Muir et al., 1985a
1,3,7,8-tetraCDD $C_{12}H_4Cl_4O_2$	50585-46-1	321.96	N/A	193.5-195	N/A	N/A	N/A	Kende et al., 1974; NIOSH, 1983
2,3,6,7-tetraCDD $C_{12}H_4Cl_4O_2$	34816-53-0	321.96	N/A	N/A	N/A	N/A	N/A	NIOSH, 1983
2,3,7,8-tetraCDD $C_{12}H_4Cl_4O_2$	1746-01-6	321.96	Colorless Needles, Crystalline	305-306	1.7×10^{-6} to 1.5×10^{-9}	6.15	0.2	Pohland and Yang, 1972 U.S. EPA, 1978, 1984a, 1985b; NIOSH, 1983; Esposito et al., 1980
pentaCDD (isomer unspecified) $C_{12}H_3Cl_5O_2$	N/A	356.4	N/A	N/A	N/A	6.8 at 25^{o}C	.04 at 25^{o}C	U.S. EPA, 1985b
1,2,3,4,7-pentaCDD $C_{12}H_3Cl_5O_2$	N/A	356.4	Colorless Solid	195-196	N/A	8.64-9.7	.132	Pohland and Yang, 1972; Sarna et al., 1984; NIOSH, 1983; Muir et al., 1985a
1,2,3,7,8-pentaCDD $C_{12}H_3Cl_5O_2$	40321-76-4	356.4	N/A	240-241	N/A	N/A	N/A	NIOSH, 1983 Gray et al., 1976

TABLE A.1 (continued)

PHYSICAL AND CHEMICAL CHARACTERISTICS OF CDDs AND CDFs

Compound/ Molecular Formula	CAS Number	Molecular Weight (grams/ mole)	Physical State (at Standard Temperature(^{o}C) and Pressure)	Melting Point	Vapor Pressure (mmHg)	Log K_{ow} (Octanol/ Water Partition Coefficient[a])	Solubility in Water (ug/l)	Reference
Page 3								
CDDs								
1,2,4,7,8-pentaCDD $C_{12}H_3Cl_5O_2$	58802-08-7	356.4	N/A	N/A	N/A	N/A	N/A	NIOSH, 1983; Pohland and Yang, 1972
hexaCDD (isomer unspecified) $C_{12}H_2Cl_6O_2$	N/A	N/A	N/A	N/A	N/A	7.6 at 25^{o}C	.008 at 25^{o}C	U.S. EPA, 1985b
1,2,3,4,7,8-hexaCDD $C_{12}H_2Cl_6O_2$	39227-28-6	390.84	Colorless Solid	275	N/A	9.19 10.5	N/A	Pohland and Yang, 1972; Sarna et al., 1984; NIOSH, 1983; Muir et al., 1985a
1,2,3,6,7,8-hexaCDD $C_{12}H_2Cl_6O_2$	34465-46-8	390.84	N/A	285-286	N/A	N/A	N/A	Gray et al., 1976; NIOSH, 1983
1,2,3,6,7,9-hexaCDD $C_{12}H_2Cl_6O_2$	N/A	390.84	N/A	N/A	N/A	N/A	N/A	Pohland and Yang, 1972;
1,2,3,7,8,9-hexaCDD $C_{12}H_2Cl_6O_2$	19408-74-3	390.84	N/A	243-244	N/A	N/A	N/A	Gray et al., 1975; NIOSH, 1983
1,2,4,6,7,9-hexaCDD $C_{12}H_2Cl_6O_2$	N/A	390.84	Colorless Solid	238-240	6.6×10^{-7}	N/A	N/A	Pohland and Yang, 1972; U.S. EPA, 1978
1,2,3,4,6,7,8-heptaCDD $C_{12}HCl_7O_2$	35822-46-9	390.84	N/A	N/A	N/A	11.5	N/A	NIOSH, 1983; Sarna et al., 1984
1,2,3,4,6,7,9-heptaCDD $C_{12}HCl_7O_2$	N/A	390.84	N/A	N/A	3.0×10^{-7}	N/A	N/A	NIOSH, 1983; U.S. EPA, 1978

TABLE A.1 (continued)

PHYSICAL AND CHEMICAL CHARACTERISTICS OF CDDs AND CDFs

Compound/ Molecular Formula	CAS Number	Molecular Weight (grams/ mole)	Physical State (at Standard Temperature(^{o}C) and Pressure)	Melting Point	Vapor Pressure (mmHg)	Log K_{ow} (Octanol/ Water Partition Coefficient[a])	Solubility in Water (ug/l)	Reference
CDDs								
octaCDD $C_{12}Cl_8O_2$	3268-87-9	459.72	N/A	330	1.8×10^{-7}	10.07 12.6	.0004	NIOSH, 1983; Pohland and Yang, 1972; Muir et al., 1985a; U.S. EPA, 1978
CDFs[c]								
monoCDF $C_{12}H_7C_{10}$	N/A	202.42	N/A	N/A	N/A	N/A	N/A	Sarna et al., 1984
diCDF $C_{12}H_6Cl_2O$	43047-99-0	237.08	N/A	N/A	7.0×10^{-6}	N/A	N/A	NIOSH, 1983; U.S. EPA, 1978
2,3,7,8-tetraCDF $C_{12}H_4Cl_4O$	51207-31-9	305.96	N/A	N/A	2.0×10^{-6}	N/A	N/A	NIOSH, 1983; U.S. EPA, 1978

a) All K_{ow} values are averaged values of experimentally derived quantities using reversed-phase HPLC within each laboratory.

b) 1,3,6,8-tetraCDD Henry's Constant is 6.81×10^{-5} atm $m^3/^{o}k$ mol (Pohland and Yang, 1972).

c) Information concerning CDFs is very limited.

d) N/A = "not available".

Freeman and Schroy, 1986), 5.6×10^{-7} (25°C) (Podoll, 1986) and 1.7×10^{-6} (estimated at 25°C) (U.S. EPA, 1978). These are low vapor pressures, generally representing low volatility. However, Freeman and Schroy (1986) noted that DDT, which has a similar vapor pressure of 1.4×10^{-7} is known to volatilize readily from soil and water.

LOG K_{OW} (LOGARITHM OF OCTANOL/WATER PARTITION COEFFICIENT)

This coefficient measures the partitioning of a compound into the two phases of an octanol-water mixture, indicating the compound's relative concentration in these two solvents. The partition coefficients of CDDs and CDFs are relatively high (i.e., the compounds preferentially dissolve in octanol and lipid) and generally increase with increasing chlorination. In lieu of actual field data, the partition coefficients are frequently used as indicators for potential concentration in biota.

SOLUBILITY IN WATER AND OTHER SOLVENTS

In general, CDDs and CDFs have extremely low water solubilities, are only slightly soluble in most organic solvents such as acetone, but are more soluble in others. The available data suggest that the less chlorinated compounds (i.e., diCDD and triCDD) are more soluble in aliphatic solvents (i.e., acetone, methanol) whereas the more highly chlorinated compounds are more soluble in aromatic hydrocarbon solvents. CDDs and CDFs are insoluble in dilute alkali, although the more highly chlorinated compounds (i.e., heptaCDD and octaCDD) are "degraded" by a few minutes' boiling with aqueous-alcoholic potassium hydroxide (Crosby, 1981). The solubility of 2,3,7,8-tetraCDD and octaCDD in various solvents is shown in Table A.2.

TABLE A.2

SOLUBILITY OF 2,3,7,8-TETRACDD AND OCTACDD IN VARIOUS SOLVENTS AT 25°C

Solvent	Solubility (mg/l) 2,3,7,8-tetraCCD	octaCDD
o-dichlorobenzene	1400	1830
chlorobenzene	720	---
anisole	---	1730
xylene	---	3580
benzene	570	---
chloroform	370	560
n-octanol	48	---
methanol	10	---
acetone	11	---
dioxane	---	380
water	0.0002	---

Source: Esposito et al., 1980

APPENDIX B

SOURCES OF CDDs AND CDFs

APPENDIX B

SOURCES OF CDDs AND CDFs

The CDD and CDF toxicity has been brought to public attention through media coverage of several major incidents: the chemical plant accident in Seveso, Italy in 1976; the fire in the Binghamton, N.Y. State Office Building in 1981; the poisonings at horse arenas in Missouri in 1971 and in Times Beach, MO, in 1982-83; the Yusho disease in Japan in 1968 and in Taiwan in 1979; and the herbicide spraying program in Vietnam in the late 1960's (Rappe, 1984).

CDDs and CDFs are not intentionally produced, except for the synthesis of analytical standards. Rather, they are found as impurities in a variety of commercial products like chlorinated phenols and their derivatives, chlorinated diphenyl ethers and polychlorinated biphenyls. They are also formed through the combustion of certain chlorinated hydrocarbons. A description of these and other sources of CDDs and CDFs follows:

PHENOXY HERBICIDES

Concentrations of .02 to 54 ug/g of 2,3,7,8-tetraCDD have been found in drums of the phenoxy herbicide 2,4,5-trichlorophenoxy acetic acid (more commonly called Agent Orange or 2,4,5-T) (Firestone, 1978; Esposito et al., 1980).

The parent compound of 2,4,5-T is 2,4,5-trichlorophenol. From 2,4,5-trichlorophenol, several other herbicides, including Silvex, are derived (U. S. EPA, 1985c). It should be pointed out that the occurrence of 2,3,7,8-tetraCDD in the environment can be mainly related to the synthesis of 2,4,5-trichlorophenol and the use of products prepared from this compound or incineration reactions. The occurrence of other CDDs and CDFs can be related to the synthesis and use of a variety of other products (WHO, 1985).

HEXACHLOROPHENE

The bactericide, hexachlorophene, also is prepared from 2,4,5-trichlorophenol. Samples from one study showed concentrations of 0.2 to 0.5 ug/g of 2,3,7,8-tetraCDD (Baughman, 1974).

CHLOROPHENOLS

Chlorophenols are commercially made either by direct chlorination of phenol or by hydrolysis of chlorobenzenes, with the process dependent on the compound desired. Chlorination of phenols yields 2,4-dichlorophenol, 2,4,6-trichlorophenol, 2,3,4,6-tetrachlorophenol, or pentachlorophenol (PCP). Hydrolysis of chlorobenzenes is used mainly for the production of 2,4,5-trichlorophenol and PCP (Nilsson, et al., 1978).

Chlorophenols have been used extensively in the wood industry as fungicides, bactericides, slimicides, and mold inhibitors. The most important use of 2,4,6-tri, 2,3,4,6-tetra, and pentachlorophenols (or their salts) is for wood protection and preservation against fungal damage. Chlorophenols contain a variety of contaminants including CDDs and CDFs, as in Table B.1 following:

TABLE B.1

CDD AND CDF CONCENTRATIONS IN COMMERCIAL CHLOROPHENOLS (ug/g)
(RAPPE et al., 1979)

	2,4,6-trichloro-phenol	2,3,4,6-tetrachloro-phenol	PCP[a/]	PCP
TetraCDDs	<0.1	<0.1	<0.1	<0.1
PentaCDDs	<0.1	<0.1	<0.1	<0.1
HexaCDDs	<1	<1	<1	2.5
HeptaCDDs	<1	10	0.5	175
OctaCDD	<1	2	4.3	500
TetraCDFs	1.5	0.5	<0.1	<0.1
PentaCDFs	17.5	10	<0.1	<0.1
HexaCDFs	36	70	0.03	<0.3
HeptaCDFs	4.8	70	0.5	19
OctaCDF	<1	10	1.1	25

[a/] Purified product

POLYCHLORINATED BIPHENYLS (PCBs)

Bowes et al., (1975), examined PCB formulations produced in the United States (Aroclor), France (Phenoclor), and Germany (Clophen). They reported that the most abundant CDFs had the same retention time as 2,3,7,8-tetraCDF and 2,3,4,7,8-pentaCDF. Quantitative results, reported as isomer group concentrations of CDFs in commercial PCBs, are given in Table B.2.

TABLE B.2

CDF CONCENTRATIONS IN COMMERCIAL PCBs (ug/g)
(Bowes et al., 1975)

Sample	Tetra	Penta	Hexa	Total
Aroclor 1248 (1969)	0.5	1.2	0.3	2.0
Aroclor 1254 (1969)	0.1	0.2	1.4	1.7
Aroclor 1254 (1970)	0.2	0.4	0.9	1.5
Aroclor 1260 (1969)	0.1	0.4	0.5	1.0
Aroclor 1260 (lot AK3)	0.2	0.3	0.3	0.8
Aroclor 1016 (1972)	ND	ND	ND	--
Clophen A 60	1.4	5.0	2.2	8.4
Phenoclor DP-6	0.7	10.0	2.9	13.6

ND = Not detected

DIPHENYL ETHER HERBICIDES

Yamagishi, et al. (1981) found CDDs and CDFs in the commercial diphenyl ether herbicides, CNP, NIP, and X-52. The major tetraCDDs identified were 1,3,6,8 and 1,3,7,9-isomers. No 2,3,7,8-tetraCDD could be found in these samples. Table B.3 summarizes the results of this study:

TABLE B.3

CDD AND CDF CONCENTRATIONS IN COMMERCIAL
DIPHENYL ETHER HERBICIDES (ug/g)

(Yamagishi et al., 1981)

	CNP	NIP	X-52
TriCCDs	ND	0.15	0.03
TetraCDDs	14.0	0.38	0.03
PentaCDDs	37	0.05	0.01
HexaCDDs	0.8	ND	ND
MonoCDFs	ND	0.34	0.48
DiCDFs	0.35	0.12	0.21
TriCDFs	0.41	0.47	0.45
TetraCDFs	0.4	0.29	0.32
PentaCDFs	1.0	ND	0.08
HexaCDFs	0.2	ND	ND

ND = Not detected

FLY ASH AND COMBUSTION PRODUCTS

Combustion sources of CDDs and CDFs have only been studied for a relatively short period of time. It is believed that CDDs and CDFs adsorb onto airborne particles which are eventually deposited on soil and water. According to a recent California Air Resources Board (CARB) report (CARB and CDHS, 1986), emissions of CDDs and CDFs from combustion sources into the atmosphere appear to be the major environmental source of dioxins. Few potential sources, except for municipal waste resource recovery facilities, have been adequately tested. Based on tests of municipal waste resource recovery facilities of the type expected to be built in California, CARB estimates that 18 to 308 pounds of CDDs and 41 to 663 pounds of CDFs would be emitted in California annually if all currently proposed facilities (39) are constructed (CARB, 1986). Table B.4 identifies potential CDD and CDF sources in California:

TABLE B.4

POTENTIAL CDD AND CDF SOURCES IN CALIFORNIA
(CARB AND CDHS, 1986)

Source Category	Operational in Calif.	Proposed for Calif.	Estimate of Relative Emissions[1]
Point Sources:			
Municipal Waste Incinerators and Refuse Derived Boilers	1	35	High
Commercial Waste Oil Burners	30+	ND	Unknown
Hazardous Waste Incinerators	17	3	Low
Industrial Boilers Cofiring Wastes	0	0	Unknown
Wire Reclamation Incinerators	76[2]	ND	Unknown
Sewage Sludge Incinerators	8	ND	Unknown
Wood/Bark Boilers	59	ND	High[3]
Black Liquor Boiler	4	0	Unknown
PCP Sludge Incinerators	ND	ND	High
Cement Kilns Cofiring Wastes	1	1	Low
Hospital Incinerators	311[2]	ND	Unknown
Sawmills[4]	86	ND	High[3]

TABLE B.4 (continued)

POTENTIAL CDD AND CDF SOURCES IN CALIFORNIA
(CARB AND CDHS, 1986)

Source Category	Operational in Calif.	Proposed for Calif.	Estimate of Relative Emissions[1/]
Area Sources:			
Mobile Sources	NA		Unknown
Wood Stove/Fireplaces	NA		Unknown
Forest Fire/Agricultural Burning	NA		Unknown

ND - no data
NA - not applicable

1/ This is an estimate of the expected emissions relative to the other sources listed.

2/ Statewide number estimated from data supplied by San Diego Air Pollution Control District and the South Coast Air Quality Management District.

3/ When burning wood treated with chlorophenol; otherwise sawmills are rated as "low".

4/ Most sawmills have the capability to incinerate some or all of the woodwaste produced at the facility. A wood/bark boiler may be used at a a sawmill to incinerate wastes. This source category may overlap other source categories listed in the table.

Combustion sources believed to have the greatest potential to emit CDDs have been identified by U.S. EPA (1984b) and are presented in Tables B.5 and B.6.

TABLE B.5

COMBUSTION SOURCES BELIEVED TO HAVE THE GREATEST POTENTIAL TO EMIT CDDs (U.S. EPA, 1984b)

Source	Rationale
Municipal Waste Incinerators	TetraCDD[1/] Detected
Refuse Derived Fuel[2/] Boilers	TetraCDD Detected
Commercial Waste Oil Burners	TetraCDD Detected
Hazardous Waste Incinerators	TetraCDD Detected
Industrial Boilers Cofiring Wastes	TetraCDD Detected
Wire Reclamation Incinerators	TetraCDD Detected
Pentachlorophenol Sludge Incinerators	TetraCDD Detected
Sewage Sludge Incinerators	CDD[2/] Detected
Mobile Sources	TetraCDD Detected
Wood Stove/Fireplaces	TetraCDD Detected
Wood/Bark Boilers	Experimental results with pentachlorophenol-treated wood
Black Liquor Boilers	Elevated polycyclic organic matter in effluent
Cement/Lime Kilns Cofiring Wastes	Precursors present
Hospital Incinerators	Burn plastics, equipped with low stacks and are located in urban areas
Forest/Grass/Agricultural Burning	Areas where chlorinated pesticides have been applied

1/ TetraCDD = tetrachlorodibenzodioxin. Available analyses are mixed, with some researchers reporting "total tetras" and others reporting 2,3,7,8-tetraCDD or both. The presence of tetraCDDs generally indicates some likelihood of 2,3,7,8-tetraCDD being present.

2/ CDD = Total of all chlorinated dibenzodioxin congeners. While detection of CDDs does not necessarily indicate presence of tetraCDD or 2,3,7,8-tetraCDD, there are sufficient data to infer such in this case.

TABLE B.6

FORMATION OF CDDs AND CDFs BY THERMAL PROCESSES
(Rappe, 1984)

Starting Material	Thermal Process	Product
2,4,5-T salt	Pyrolysis	2,3,7,8,-tetraCDD
2,4,5-T (on vegetation)	Pyrolysis	No tetraCDD
	Burning	No tetraCDD
Chlorophenate	Burning	CDDs[a] + CDFs
Polychlorinated biphenyls	Pyrolysis	CDFs[b]
Polychlorobenzenes	Pyrolysis	CDFs + CDDs[c]
Chlorodiphenyl ethers	Pyrolysis	CDFs + CDDs
Polyvinylchloride	Pyrolysis	Polychlorobenzenes

[a] CDDs formed by dimerization and a nonspecific dechlorination

[b] Other products: hexa- and pentachlorobenzenes

[c] Other products: PCBs, polychlorinated naphthalenes

APPENDIX D

WORLDWIDE DETECTION OF CDDs AND CDFs

APPENDIX D

WORLDWIDE DETECTION OF CDDs AND CDFs

Over the past 10 years, extensive data have been collected on both sources and levels of CDDs and CDFs in the environment. While 2,3,7,8-tetraCDD was the initial congener of concern and most of the earlier results report only this compound, the realization that other congeners chlorinated at the 2,3,7, and 8 positions also possess significant toxicity has lead to greater efforts to search for them in a variety of environmental compartments.

This appendix presents an overview of significant CDD and CDF contamination incidents worldwide, as well as a limited number of examples where these compounds were detected in California.

ENVIRONMENTAL CONTAMINATION--WORLDWIDE

This section summarizes reported worldwide incidents, excluding California, where releases of CDDs and CDFs into the environment have occurred. (California data are discussed separately in this appendix). Ranges of representative worldwide values of CDD and CDF isomer groups or individual congeners are presented in four separate tables.

Table D.1 shows concentrations of the CDD and CDF isomer groups found in a variety of commercial products. Table D.2 provides ranges of concentrations reported in water, soil, sediment and air. Table D.3 presents reported values found in biota (fish, animals, plants) and Table D.4 lists reported ranges of specific congeners detected in humans (adipose, liver, blood, milk). For each table there is a discussion of the major incidents which led to the CDD or CDF exposure.

Products Containing CDDs and CDFs--Table D.1

The CDDs and CDFs are not intentionally produced, but are found as impurities in a variety of commercial products such as wood preservatives (chlorinated phenols), phenoxy herbicides, PCBs and diphenyl ether herbicides. The amount of these impurities depends upon the method of preparation. U.S. EPA (1978), Da Roos et al., (1981), Rappe (1984) and Buser et al. (1976) have reported the results of some typical analyses of commercial products containing CDDs and CDFs.

Firestone (1978) and U.S. EPA (Esposito et al., 1980) have found tetraCDD in the ppt range in drums of 2,4,5-trichlorophenoxy acetic acid (2,4,5-T, a component of Agent Orange). Earlier, in the 1960's, the mean level of 2,3,7,8-tetraCDD in Agent Orange preparations was 1.98 ppm (U.S. EPA, 1985b). At the present time, producers claim that their products contain less than 0.1 ppt of 2,3,7,8-tetraCDD (U.S. EPA, 1985b).

APPENDIX D

TABLE D.1

CDD AND CDF CONCENTRATIONS (ppm) IN COMMERCIAL PRODUCTS

Description	Tetra CDD	Penta CDD	Hexa CDD	Hepta CDD	Octa CDD	Tetra CDF	Penta CDF	Hexa CDF	Hepta CDF	Octa CDF
Wood Preservatives										
Wood Treatment Oil containing 4.5% PCP (2,3,7,8-specific) (Da Roos et al., 1981)	0.001	0.033	0.574	0.256	3.99	0.018	0.137	1.813	0.114	0.711
Unpurified Commercial PCP (Rappe, 1984)	<0.1	<0.1	2.5	175	500	<0.1	<0.1	<0.3	19	25
Unpurified Commercial PCP (88% Penta) (U.S. EPA, 1978)	NA	NA	4	125	2500	NA	NA	30	80	80
Technical Grade PCP Reduced by Distillation (89% Penta) (U.S. EPA, 1978)	NA	NA	1.0	6.5	15	NA	NA	1.0	1.8	1.0
Unpurified Commercial PCP flakes (Buser and Bosshardt, 1976)	<0.02	<0.03	5.2	95	280	0.02	0.40	28	200	230
Unpurified Commercial PCP-Na Powder (Buser and Bosshardt, 1976)	0.16	0.03	<0.03	0.3	1.2	<0.02	<0.03	0.20	1.2	3.0

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TABLE D.1 (continued)

CDD AND CDF CONCENTRATIONS (ppm) IN COMMERCIAL PRODUCTS

Description	Tetra CDD	Penta CDD	Hexa CDD	Hepta CDD	Octa CDD	Tetra CDF	Penta CDF	Hexa CDF	Hepta CDF	Octa CDF
Unpurified Commercial PCP-Na Pellets (Buser and Bosshardt, 1976)	0.08	0.03	0.25	2.8	5.1	0.02	0.13	4.1	13	8.6
Unpurified Commercial PCP-Na Granules (Buser and Bosshardt, 1976)	0.05	<0.03	3.4	40	115	<0.02	0.05	11	50	24
Phenoxy Herbicides										
2,4,5-T (acids, esters, & formulated products) (2,3,7,8-specific) (U.S. EPA, 1985b)	0.01-0.08	NA	NA	NA	NA	NA	NA	NA	NA	NA
Agent Orange (mixture of 2,4,5-T & 2,4-D) mean value (Firestone, 1978; Esposito et al., 1980)	.002-.054	NA	NA	NA	NA	NA	NA	NA	NA	NA
Commercial PCB										
Aroclor 1248 (Rappe, 1984)	NA	NA	NA	NA	NA	0.5	1.2	0.3	NA	NA
Clophen A-60 (Rappe, 1984)	NA	NA	NA	NA	NA	1.4	5.0	2.2	NA	NA
Phenoclor DP-6 (Rappe, 1984)	NA	NA	NA	NA	NA	0.7	10.0	2.9	NA	NA

APPENDIX D

TABLE D.1 (continued)

CDD AND CDF CONCENTRATIONS (ppm) IN COMMERCIAL PRODUCTS

Description	Tetra CDD	Penta CDD	Hexa CDD	Hepta CDD	Octa CDD	Tetra CDF	Penta CDF	Hexa CDF	Hepta CDF	Octa CDF
Diphenyl Ether Herbicides										
CNP (Yamagashi et al., 1981)	14.0	37	0.8	NA	NA	0.4	1.0	0.2	NA	NA
NIP (Yamagashi et al., 1981)	0.38	0.05	ND	NA	NA	0.29	ND	ND	NA	NA
X-52 (Yamagashi et al., 1981)	0.03	0.01	ND	NA	NA	0.32	0.08	ND	NA	NA

ND - Not Detected
NA - Not Available

Rappe (1984) examined a series of commercial PCBs of both United States and European manufacture. He reported that the most abundant CDFs had the same retention time as 2,3,7,8-tetraCDF and 2,3,4,7,8-pentaCDF.

Yamagishi et al. (1981) found levels of both CDDs and CDFs in preparations of the commercial diphenyl ether herbicides CNP, NIP, and X-52. The major tetraCDDs identified were 1,3,6,8 and 1,3,7,9 isomers. The 2,3,7,8-tetraCDD isomer was not found in these samples.

CDDs and CDFs in Water, Soil, Sediment and Air--Table D.2

Thibodeaux (1983) assessed quantities of 2,3,7,8-tetraCDD in surface water, soil, creek and pond sediment resulting from an herbicide production facility in Jacksonville, Arkansas that practiced onsite disposal. The waste was stored in metal drums buried onsite or deposited on soil or into water bodies within the plant boundary. The plant had been manufacturing 2,4-D and 2,4,5-T since 1958. 2,3,7,8-TetraCDD was not detected until spring 1979, after which approximately 1000 soil, air, water, and sediment samples were taken by government and company representatives. The pond sediment from onsite appeared to be the most contaminated source of 2,3,7,8-tetraCDD (22.1 $\pm$ 2.1 ppb 2,3,7,8-tetraCDD).

Pereira et al. (1985) performed geochemical investigations of pond sludge, groundwater and porous media from the unsaturated and saturated zones at a wood treatment facility in Pensacola, Florida in July 1983. The facility had been discharging creosote and PCP into two unlined surface impoundments on site, resulting in contamination of the underlying sand and gravel aquifer. It had operated from 1902 to 1981 but has since discontinued all operations. Researchers found that CDDs had migrated both vertically and horizontally in the subsurface and were present at considerable distances from the source of contamination. Ground water samples taken at various depths were generally in the ppt range (wet weight) whereas concentrations of CDDs in the porous media of the saturated and unsaturated zones were in the ppb range (dry weight). Significant concentrations of hexaCDD, heptaCDD, and octaCDD associated with the sediment and bottom material of two surface impoundments were reported in the ppm range (wet weight).

In Missouri, horse arenas were sprayed in 1971 with 2,000 gallons of a dust control solution made from trichlorophenol (triCP) distillation products mixed with motor oil. Subsequently, animals living on or near the arenas died and several children became ill. Sampling of the soil in the arenas between 1971 and 1972 indicated very high (ppm) concentrations of tetraCDD, triCP, and PCBs. After the soil was excavated twice from one arena in 1974, no detectable concentrations of tetraCDD or PCB and only trace amounts of triCP were found (Reggiani, 1980). Eight years

APPENDIX D

TABLE D.2

CDD AND CDF CONCENTRATIONS (ppb) IN WATER, SOIL, SEDIMENT, AND AIR

Description		Tetra CDD	Penta CDD	Hexa CDD	Hepta CDD	Octa CDD	Tetra CDF	Penta CDF	Hexa CDF	Hepta CDF	Octa CDF
Wastewater											
Surface Water at a 2,4-D & 2,4,5-T production facility in Jacksonville, Arkansas (2,3,7,8-Specific) (Thibodeaux, 1983)		0.014	NA	NA	NA	NA	NA	NA	NA	NA	NA
Waste water from a wood preservation plant in Ottawa, Canada (Lao et al., 1983)		0.0012	0.0083	0.034	0.122	0.258	0.0007	0.0015	0.0115	0.048	0.038
Rainwater runoff sump sludge at a wood preservation plant (site unidentified) (U.S. EPA, 1986f)		ND	ND	<4.5	111	262	ND	ND	ND	<2.3	23.6
Ambient Water											
Perched ground water (oil sheen filtered) (site unidentified) (U.S. EPA, 1986f)		ND	ND	0.31	5.8	50	ND	ND	722	6000	23000
	Ft.										
Ground water from an abandoned wood treatment facility in Pensacola, Florida - depth to water in feet. (Pereira et al., 1985)	20	NA	NA	0.061	1.500	3.900	NA	NA	NA	NA	NA
	40	NA	NA	0.0019	0.0046	0.0217	NA	NA	NA	NA	NA
	60	NA	NA	0.021	0.034	0.039	NA	NA	NA	NA	NA
	80	NA	NA	ND	ND	ND	NA	NA	NA	NA	NA
	100	NA	NA	ND	0.0004	0.0014	NA	NA	NA	NA	NA

APPENDIX D

TABLE D.2 (continued)

CDD AND CDF CONCENTRATIONS (ppb) IN WATER, SOIL, SEDIMENT, AND AIR

Description	Tetra CDD	Penta CDD	Hexa CDD	Hepta CDD	Octa CDD	Tetra CDF	Penta CDF	Hexa CDF	Hepta CDF	Octa CDF
Soil										
Surface soil (upper 7 cm) from Seveso, Italy after industrial accident. Soil density 1.4 kg/liter (Reggiani, 1980)										
Highest value closest to factory	55	NA	NA	NA	NA	NA	NA	NA	NA	NA
Highest value in formerly inhabited area	20	NA	NA	NA	NA	NA	NA	NA	NA	NA
Limit for evacuation	0.1	NA	NA	NA	NA	NA	NA	NA	NA	NA
Missouri horse arenas that were sprayed for dust control using TCDD contaminated industrial waste residues (Reggiani, 1980)										
Moscow Mills Arena in Lincoln County, August, 1971	31,800-33,000	NA	NA	NA	NA	NA	NA	NA	NA	NA
New Bloomfield Arena in Phelps County,	220-850	NA	NA	NA	NA	NA	NA	NA	NA	NA
St. James Arena in Calloway County, August, 1974	120	NA	NA	NA	NA	NA	NA	NA	NA	NA

APPENDIX D

TABLE D.2 (continued)

CDD AND CDF CONCENTRATIONS (ppb) IN WATER, SOIL, SEDIMENT, AND AIR

Description	Tetra CDD	Penta CDD	Hexa CDD	Hepta CDD	Octa CDD	Tetra CDF	Penta CDF	Hexa CDF	Hepta CDF	Octa CDF
Soil										
Surface soil from on-site disposal of 2,4-D and 2,4,5-T in Jacksonville, Arkansas (2,3,7,8-specific) 5 samples (Thibodeaux, 1983)										
Average	1.3	NA	NA	NA	NA	NA	NA	NA	NA	NA
Range	ND to 2.9	NA	NA	NA	NA	NA	NA	NA	NA	NA
Surface soil from Times Beach, Missouri. Samples taken in early December 1982 highest concentration detected of 2,3,7,8-tetraCDD) (Kleopfer, 1985)	1200	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sediment										
On-site disposal of 2,4,D and 2,4,5-T in Jacksonville, Arkansas (2,3,7,8-Specific) (Thibodeaux, 1983)										
Creek Sediment (average of 5 samples)	.77	NA	NA	NA	NA	NA	NA	NA	NA	NA
Range	ND to 1.8	NA	NA	NA	NA	NA	NA	NA	NA	NA
Pond Sediment (average of 3 samples)	22.1	NA	NA	NA	NA	NA	NA	NA	NA	NA
Range	±2.1	NA	NA	NA	NA	NA	NA	NA	NA	NA

APPENDIX D

TABLE D.2 (continued)

CDD AND CDF CONCENTRATIONS (ppb) IN WATER, SOIL, SEDIMENT, AND AIR

Description	Tetra CDD	Penta CDD	Hexa CDD	Hepta CDD	Octa CDD	Tetra CDF	Penta CDF	Hexa CDF	Hepta CDF	Octa CDF
Sediment										
From Swiss Lakes near municipal incinerators (dry weight) (average of 3 labs)(Czuczwa et al., 1985)	ND	.05	.13	.35	1.3	.08	ND	.02	.20	.15
Samples from a refuse dump near Amsterdam, Holland (dry weight) 20% organic content (Heida, 1983)										
- within dump area (range)	.844-5.062	NA	NA	NA	NA	NA	NA	NA	NA	NA
- outside dump area (range)	.055-.611	NA	NA	NA	NA	NA	NA	NA	NA	NA
From a wood treatment facility in Pensacola, Florida (average of 2 samples) (Pereira et al., 1985)	NA	NA	373	10,750	94,000	NA	NA	NA	NA	NA
Sludge										
Pond sludge from a wood treatment facility in Pensacola, Florida (average of 2 samples) (Pereira et al., 1985)	NA	NA	365	9,020	39,500	NA	NA	NA	NA	
Dust										
Samples from Dow Chemical Research Building in Midlands, Michigan (Esposito et al., 1980)	.5-2.3	NA	9-35	140-1200	650-7500	NA	NA	NA	NA	NA

APPENDIX D

TABLE D.2 (continued)

CDD AND CDF CONCENTRATIONS (ppb) IN WATER, SOIL, SEDIMENT, AND AIR

Description	Tetra CDD	Penta CDD	Hexa CDD	Hepta CDD	Octa CDD	Tetra CDF	Penta CDF	Hexa CDF	Hepta CDF	Octa CDF
Soot										
From Binghamton, State Office Building. Each Lab received samples collected at different times and locations (Schecter et al., 1985b)										
Lab 1	<3,000	<2,000	<3,000	7,000	5,000	1.92×10^6	1.2×10^6	1.16×10^6	4.05×10^5	66,000
Lab 2	1,200	5,000	5,000	7,000	2,000	28,000	6.7×10^5	9.65×10^5	4.6×10^5	40,000
Air										
Fly Ash from a municipal incinerator in Switzerland (NRCC, 1984)	NA	NA	NA	NA	NA	1.0	4.0	30.0	40.0	10.0
Atmospheric dust from Seveso, Italy after industrial accident (2,3,7,8-Specific) (U.S. EPA, 1985b)	.06-.50	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air particulate sample from a waste disposal site near Jacksonville, Arkansas (average concentration of tetraCDD) (U.S. EPA, 1985b)	1.1	NA	NA	NA	NA	NA	NA	NA	NA	NA

APPENDIX D

TABLE D.2 (continued)

CDD AND CDF CONCENTRATIONS (ppb) IN WATER, SOIL, SEDIMENT, AND AIR

Description	Tetra CDD	Penta CDD	Hexa CDD	Hepta CDD	Octa CDD	Tetra CDF	Penta CDF	Hexa CDF	Hepta CDF	Octa CDF
Air										
Airborne particulates from municipal incinerator sample over a 5-hour period during which 5.8 m^3 of stack gas was released at the Hampstead Refuse Recovery Corporation, Nassau County, New York (Tiernan et al., 1983)	.38	.53	.85	2.00	.49	2.60	1.60	1.80	2.20	.17
Airborne particulates from Hamilton Municipal Incinerator, Ontario, Canada, 1983-1984 (average of 3 tests) (NRCC, 1984)	1.70	1.08	.48	.33	.12	5.64	3.03	.47	.15	.05

NA = Not available
ND = Not detected (values in parentheses are limits of detection)

later the horse arenas were reinvestigated (Kleopfer, 1985). As of August 1982, 10,734 Missouri soil samples had been processed for analysis of 2,3,7,8-tetraCDD and 92 percent of the results of these analyses had been validated through a quality assurance plan. Of the validated results, 23 percent were positive and above 1.0 ppb, while 2.2 percent were above 100 ppb (identity of excavated vs. non-excavated sites was not given).

In 1981 a fire involving an electrical transformer containing a mixture of PCBs and chlorinated benzenes occurred at a Binghamton, New York office building. This led to contamination of the building structure with CDF and CDD laden soot. Various samples revealed very high concentrations of the CDDs (ppm range) and CDFs (parts per thousand range) (Schecter et al., 1985b). Two years after several cleanup operations, air samples still showed measurable values for selected isomer groups.

Heida (1983) examined tetraCDD in sediment from ponds and canals located near a refuse dump in Amsterdam, Holland. The 2,3,7,8-tetraCDD concentrations originated from 2,4,5-T production. The results showed that the highest concentration of 2,3,7,8-tetraCDD (5.0 ppb) occurred in the main drainage canal near the dump and rapidly decreased outside the dump area. Analysis of eel flesh revealed only two samples (total samples not given) from the sampling site inside the dump area which contained small quantities of 2,3,7,8-tetraCDD (3.9 ppb in body fat).

In 1972, and again in 1973, the streets of Times Beach, Missouri were oiled by a firm specializing in waste oil reclamation. Mixed with the waste oil were impurities ("still-bottoms") from the production of 2,4,5-trichlorophenol (Kleopfer, 1985). The soil was sampled by U.S. EPA in early December 1982 (Kleopfer, 1985), and the highest level of 2,3,7,8-tetraCDD detected was 1,200 ppb in surface soil. The Centers for Disease Control concluded in a risk analysis that levels of 1 ppb or greater of 2,3,7,8-tetraCDD in residential soil represented an unreasonable risk. Subsequently, the town was evacuated and eventually bought out by the government.

One of the most well known incidents of 2,3,7,8-tetraCDD contamination occurred in Seveso, Italy in 1976. Here a plant manufacturing hexachlorophene exploded and contaminated about 700 acres adjoining the plant. Because of the high concentrations of 2,3,7,8-tetraCDD found in the soil, the Italian authorities evacuated 736 people from the area. Up to 5,000 people were believed to have been exposed from the explosion. Monitoring of Seveso soil one year after the accident showed that the highest concentrations of 2,3,7,8-tetraCDD were not present in the topmost soil layer (0.5 cm), but very often in the second (0.5-1.0 cm) or third (1.0-1.5 cm) layers (Reggiani, 1980).

CDDs and CDFs were found in annually laminated sediment from Lakes Zurich, Baldegg, and Lugano in Switzerland. OctaCDD predominated, averaging approximately 1.3 ppb. The congener distribution indicated that combustion was the source of CDDs and CDFs in these sediments (Czuczwa et al., 1985).

Combustion is now generally recognized as an important potential source of CDDs and CDFs in the environment. Efforts to determine concentrations in the air have been focused mainly on municipal solid waste incinerators and power plants from other states and Canada (NRCC, 1984; U.S. EPA, 1985b; Tiernan, 1983). The concentration levels in air depend on a host of features including feedstock burned, the facility design and operational variables. Only a few measurements of possible CDD and CDF precursor compounds in incinerator effluents have been made.

Reported CDD and CDF Concentrations in Biota--Table D.3

Kaczmar (1983) examined various species of fish in selected Michigan water systems for residues of 2,3,7,8-tetraCDD. Detectable residues of 2,3,7,8-tetraCDD ranged from 17 to 586 ppt. The significance to this particular study was that the investigator found residues in fish collected upstream of a chlorophenol manufacturing facility. The study suggests that low-level contamination (in the ppt range) of bottom feeding fish is relatively widespread in the industrialized portions of Michigan.

New York State Health Department performed congener specific determination of 2,3,7,8-tetraCDD levels in Great Lakes fish (NRCC, 1981). Of the 76 samples, 2,3,7,8-tetraCDD levels ranged from non-detectable to 162 ppt (detection limit not given). Fish sampled included small mouth bass, lake trout, white sucker, brown bullhead, rainbow trout, coho and chinook salmon, and brown trout. Lakes sampled included Lake Ontario, Lake Erie, Lake Huron, Lake Michigan, and Lake Superior. As a result of this study, New York State issued a health advisory fishing guideline that is more stringent than the FDA's or Canada's. See Chapter 7: Criteria and Standards.)

O'Keefe et al. (1984) examined whole body samples of striped bass from the lower Hudson River in New York for 2,3,7,8-tetraCDD and 2,3,7,8-tetraCDF. Concentrations of 2,3,7,8-tetraCDD ranged from 16 to 120 ppt. Striped bass from other locations (Rhode Island coastal waters and Chesapeake Bay, Maryland) had less than 5 ppt. Concentrations of 2,3,7,8-tetraCDF found in striped bass from all three locations ranged from 6 ppt in Chesapeake Bay to 78 ppt in the Hudson River.

APPENDIX D

TABLE D.3

2,3,7,8-TETRACDD AND TETRACDF CONCENTRATIONS (ppt) IN BIOTA

Description	2,3,7,8-tetraCDD	2,3,7,8-tetraCDF
Fish		
Carp (whole body) from Tittabawassee, below dam (1 sample) (Kaczmar, 1983)	17	NA
Carp (whole body) from St. Clair River at Decker's Landing (Lake Huron) (1 sample) (Kaczmar, 1983)	586	NA
Coho salmon (whole body) from Salmon River near Lake Ontario (1 sample) (NRCC, 1981)	31	NA
Coho salmon (whole body) from Spring Brook Weir near Lake Ontario (range of 3 composite samples) (NRCC, 1981)	19-22	NA
Coho salmon (whole body) from Belle Isle on Lake Erie (range from 3 samples) (NRCC, 1981)	0.9-1.4	NA
Coho salmon (whole body) from St. Joseph on Lake Michigan (5 samples) (NRCC, 1981)	ND (2.0-4.9)	NA
Brown trout (whole body) from St. Catherines/Niagara in Lake Ontario (1 sample) (NRCC, 1981)	162	NA
Brown trout (whole body) from Nine Mile Point in Lake Ontario (1 sample) (NRCC, 1981)	8	NA

TABLE D.3 (continued)

2,3,7,8-TETRACDD AND TETRACDF CONCENTRATIONS (ppt) IN BIOTA

Description	2,3,7,8-tetraCDD	2,3,7,8-tetraCDF
White perch (whole body) from Cape Vincent in Lake Ontario (range of 4 samples) (NRCC, 1981)	4.9-36	NA
Striped bass (whole body) from Rhode Island coastal waters (range from 5 samples) (O'Keefe et al., 1984)	2.0-5.0	17-50
Striped bass (whole body) from Little Neck Bay, Long Island, New York (range from 4 samples) (O'Keefe et al., 1984)	ND (0.3-7.1) - 39	12-22
Striped bass (whole body) from Newark Bay, New Jersey (range from 4 samples) (O'Keefe et al., 1984)	16-67	20-34
Striped bass (whole body) from Tappen Zee Bridge, Hudson River, New York (range from 4 samples) (O'Keefe et al., 1984)	ND (0.2-2.0) - 33	16-72
Striped bass (whole body) from Poughkeepsie, Hudson River, New York (2 samples) (O'Keefe et al., 1984)	120	74-78
Striped bass (whole body) Chesapeake Bay, Maryland (1 sample) (O'Keefe et al., 1984)	ND (3.5)	ND (13)

TABLE D.3 (continued)

2,3,7,8-TETRACDD AND TETRACDF CONCENTRATIONS (ppt) IN BIOTA

Description	2,3,7,8-tetraCDD	2,3,7,8-tetraCDF
Terrestrial Biota		
Field mouse (whole body) from Seveso, Italy (range of 14 samples) (Esposito et al., 1980)	70-49,000	NA
Hare (liver) from Seveso, Italy (1 sample) (Esposito et al., 1980)	7,700	NA
Toad (whole body) from Seveso, Italy (1 sample) (Esposito et al., 1980)	200	NA
Snake (liver) from Seveso, Italy (1 sample) (Esposito et al., 1980)	2,700	NA
Snake (adipose tissue) from Seveso, Italy (1 sample) (Esposito et al., 1980)	16,000	NA
Earthworm (whole body) from Seveso, Italy (average of 2 samples) (Esposito et al., 1980)	12,000	NA
Cow (milk) from Seveso, Italy (average of 9 samples) (Esposito et al., 1980)	2,196	NA

Footnotes

NA - Not Available
ND - Not Detected
Numbers in parentheses are limits of detection

several investigators (Esposito et al., 1980) have studied the levels of tetraCDD in wild animals in the contaminated area near Seveso, Italy. Field mice contained tetraCDD concentrations ranging from 70 to 49,000 ppt (mean value 4500 ppt). These mice lived on soil where the upper 7 centimeters varied from 10 to 12,000 ppt of tetraCDD (mean value 3500 ppt). Several rabbits and one snake showed tetraCDD in the liver. Liver samples from domestic birds were analyzed for tetraCDD with negative results.

CDD and CDF Concentrations in Human Tissues--Table D.4

Schecter et al. (1985b) examined human adipose tissue for tetra through octaCDD substituted congeners. All persons examined resided in upstate New York during 1983 to 1984. Tissues from exposed versus unexposed individuals were evaluated. The difference in CDD congener concentrations between the person exposed to the soot from the Binghamton State Office building transformer fire and the control group was surprisingly small for most congeners. Penta- and hexaCDFs were also found in the exposed and, to a lesser extent, in the control population. The high background contamination (in the control group) was believed by the authors to be caused by exposure to technical grade PCP or food containing PCP.

Nygren et al. (1985) performed a study to determine if there was any difference between cancer patients previously exposed to chlorinated phenoxyacetic acids over a ten year period and to unexposed controls. Adipose tissue was excised from each group. There was no difference in levels and pattern between the cancer patients and controls except for the 2,3,4,7,8-pentaCDF congener, which could not be associated with the specific exposure. The congener profile for the mean and individual values were all identical. According to the authors, the data strongly suggests that there is a background concentration of CDDs and CDFs in the general population.

The adipose tissue of Swedish workers exposed to chlorinated phenoxyacetic acids was analyzed for CDDs and CDFs and compared to adipose tissues of unexposed workers (Hardell et al., 1985). Mean levels and ranges are presented in Table D.4. Regarding CDDs, the only significant finding was hexaCDD at levels higher in exposed than in unexposed individuals. The difference was attributed to the 1,2,3,6,7,8-hexaCDD congener. Mean levels of pentaCDF and hexaCDF were significantly higher in exposed versus the unexposed individuals. No difference was found in exposed and unexposed individuals for tetraCDD.

In 1968, over 1500 persons in southwest Japan were exposed by consuming a commercial rice oil accidently contaminated by PCB, CDF and polychlorinated quaterphenyls. In 1979, a similar episode was reported in Taiwan where over 2,000 persons were exposed. These are referred to as the "Yusho" episodes.

APPENDIX D

TABLE D.4

CDD AND CDF CONCENTRATIONS (ppt) IN HUMAN TISSUE

Description	2,3,7,8-TetraCDD	Tetra CDD	1,2,3,7,8-PentaCDD	Penta CDD	1,2,3,6,7,8-HexaCDD	1,2,3,7,8,9-HexaCDD	Hexa CDD	1,2,3,4,6,7,8-HeptaCDD	Hepta CDD	Octa CDD	Total CDD
Adipose Tissue											
1 sample from Binghamton State Office building worker in New York after being exposed to CDD & CDF contaminated soot (Schecter et al., 1985a)	11.6	NA	15.0	NA	72.6	7.3	NA	209	NA	690	NA
4 people from New York that were unexposed (Schecter et al., 1985a)	3.7	NA	7.5	NA	60.4	6.8	NA	93.1	NA	586	NA
	6.0	NA	8.2	NA	60.3	7.4	NA	119	NA	695	NA
	7.2	NA	10.3	NA	54.5	7.5	NA	39.4	NA	593	NA
	8.3	NA	13.8	NA	46.2	7.4	NA	95.8	NA	534	NA
13 samples from a phenoxy acid sprayer who has been spraying for >10 years (average and standard deviation) (Nygren et al., 1985)	2±2.7	NA	6±2.5	NA	19±12	5±2.7	NA	104±93.5*	NA	98±207	NA
1 sample from exposed BASF worker who has had chloracne since 1953 (Nygren et al., 1985)	101	NA	18	NA	48	12.0	NA	20*	NA	80	NA
1 sample from chemist who has synthesized CDD and CDF isomers (Nygren et al., 1985)	NA	NA	5	NA	12	5	NA	100*	NA	374	NA
18 unexposed people (average) (Nygren et al., 1985)	3±2.0	NA	9±5.9	NA	12±3.9	4±1.0	NA	85±47*	NA	421±178	NA

APPENDIX D

TABLE D.4

CDD AND CDF CONCENTRATIONS (ppt) IN HUMAN TISSUE

Description		2,3,7,8-TetraCDD	Tetra CDD	1,2,3,7,8-PentaCDD	Penta CDD	1,2,3,6,7,8-HexaCDD	1,2,3,7,8,9-HexaCDD	Hexa CDD	1,2,3,4,6,7,8-HeptaCDD	Hepta CDD	Octa CDD	Total CDD
7 Swedish workers exposed to phenoxy acids (Hardell et al., 1985)	mean	NA	2.8	NA	11	NA	NA	31	NA	133	443	642
	range	NA	ND-9.0	NA	ND-24	NA	NA	16-68	NA	28-380	154-623	230-914
18 unexposed workers (Hardell et al., 1985)	mean	NA	2.7	NA	8.7	NA	NA	16	NA	86	426	539
	range	NA	ND-6.0	NA	ND-19	NA	NA	6-23	NA	12-176	98-679	122-879
1 Yusho baby from Taiwan (Rappe et al., 1983b)		NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
9 samples from exposed North Vietnamese 56% lipid (average) (Commoner et al., 1986)		ND	NA	0.42	NA	NA	NA	7.6	19.2	NA	92.4	NA
15 samples from exposed South Vietnamese 62% lipid (average) (Commoner et al., 1986)		22.3	NA	14.4	NA	NA	NA	99.8	178	NA	1326	NA
46 samples from exposed Americans prepared as composites from over 900 specimens 80% lipid (Commoner, et al., 1986)		6.3	NA	40	NA	NA	NA	90	110	NA	700	NA
3 samples from Vietnam veterans exposed to Agent Orange (average) (U.S. EPA 1985b)		20-173	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA

APPENDIX D

TABLE D.4

CDD AND CDF CONCENTRATIONS (ppt) IN HUMAN TISSUE

Description	2,3,7,8-TetraCDD	Tetra CDD	1,2,3,7,8-PentaCDD	Penta CDD	1,2,3,6,7,8-HexaCDD	1,2,3,7,8,9-HexaCDD	Hexa CDD	1,2,3,4,6,7,8-HeptaCDD	Hepta CDD	Octa CDD	Total CDD
1 sample from deceased Yusho patient (Masuda and and Yoshimura, 1984)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Liver Tissue											
1 deceased Yusho Patient (Masuda and Yoshimura, 1984; Rappe et al., 1983b)	2	NA	<2	NA	4	NA	NA	72	NA	350	NA
1 Yusho baby from Taiwan (Rappe et al., 1983b)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Blood											
Workers exposed to chlorophenol at a sawmill (Rappe et al., 1983b)	NA	NA	NA	NA	NA	NA	NA	<2	NA	5	NA
1 sample taken in Seveso, Italy after accident (Facchetti et al., 1980)	6	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Milk											
5 samples from South Vietnam in 1973 (average) (Commoner et al., 1986)	3	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Milk Lipid											
5 samples from South Vietnam in 1973 (average) (Commoner et al., 1986)	100	NA	NA	NA	NA	NA	NA	NA	NA	170	NA

APPENDIX D
TABLE D.4

CDD AND CDF CONCENTRATIONS (ppt) IN HUMAN TISSUE

DESCRIPTION	2,3,7,8 Tetra-CDF	Tetra-CDF	1,2,3,7,8 Penta-CDF	2,3,4,7,8 Penta-CDF	Penta-CDF	1,2,3,4,7,8 Hexa-CDF	1,2,3,6,7,8 Hexa-CDF	2,3,4,6,7,8 Hexa-CDF	Hexa-CDF	1,2,3,4,6,7,8 Hepta-CDF	1,2,3,4,7,8,9 Hepta-CDF	Hepta-CDF	Octa-CDF	Total CDF
Adipose Tissue														
1 sample from Binghampton office building worker in New York after being exposed to CDD and CDF contaminated soot (Schecter et al., 1985a)	ND(2)	NA	NA	74.7	NA	149	112	NA	NA	39.3	25.9	NA	1.6	NA
4 people from New York that were unexposed (Schecter et al., 1985a)	ND(2)	NA	NA	16.5	NA	22.9	15.4	NA	NA	23.8	20.6	NA	1.5	NA
	ND(2)	NA	NA	17.0	NA	13.0	8.8	NA	NA	12.5	19.6	NA	1.2	NA
	4.1	NA	NA	10.9	NA	9.3	5.8	NA	NA	13.7	ND	NA	ND(20)	NA
	ND(2)	NA	NA	12.5	NA	11.4	5.6	NA	NA	16.3	ND	NA	ND(20)	NA
13 samples from phenoxy acid sprayers who have been spraying for >10 years (average and standard deviation) (Nygren et al., 1985)	4±2.1	NA	NA	50±23.6	NA	7±3.8	5±23.6	2±1.8	NA	14±12.4	NA	NA	<5	NA
1 sample from exposed BASF worker who has had chloracne since 1953 (Nygren et al., 1985)	<3	NA	NA	32	NA	11	5	2	NA	37	NA	NA	<50	NA
1 sample from chemist who has synthesized CDD and CDF isomers (Nygren et al., 1985)	7	NA	NA	26	NA	12	7	38	NA	17	NA	NA	240	NA
18 unexposed people (average & S.D.) (Nygren et al., 1985)	4±2.6	NA	NA	32±14.4	NA	5±1.5	4±1.4	2±1.0	NA	10±4.6	NA	NA	<5	NA

APPENDIX D
TABLE D.4

CDD AND CDF CONCENTRATIONS (ppt) IN HUMAN TISSUE

DESCRIPTION		2,3,7,8 Tetra-CDF	Tetra-CDF	1,2,3,7,8 Penta-CDF	2,3,4,7,8 Penta-CDF	Penta-CDF	1,2,3,4,7,8 Hexa-CDF	1,2,3,6,7,8 Hexa-CDF	2,3,4,6,7,8 Hexa-CDF	Hexa-CDF	1,2,3,4,6,7,8 Hepta-CDF	1,2,3,4,7,8,9 Hepta-CDF	Hepta-CDF	Octa-CDF	Total CDF
7 Swedish workers exposed to phenoxy acids (Hardell et al., 1985)	mean	NA	4.2	NA	NA	56	NA	NA	NA	19	NA	NA	18	NA	98
	range	NA	2.1-7.2	NA	NA	22-87	NA	NA	NA	8-35	NA	NA	5-49	NA	39-17
18 unexposed workers (Hardell et al., 1985)	mean	NA	4.2	NA	NA	32	NA	NA	NA	11	NA	NA	10	NA	57
	range	NA	0.3-11.4	NA	NA	9-65	NA	NA	NA	13-17	NA	NA	1-18	NA	13-10
1 Yusho baby from Taiwan (Rappe et al., 1983b)		17	NA	44	68	NA	88	NA	NA	NA	NA	NA	NA	NA	NA
9 samples from exposed North Vietnamese (average) 56% lipid (Commoner et al., 1986)		ND	NA	NA	9.8	NA	NA	NA	NA	10.3	2.3	NA	NA	ND	142
15 samples from exposed South Vietnamese (average) 62% lipid (Commoner et al., 1986)		ND	NA	NA	21.0	NA	NA	NA	NA	58.3	28.9	NA	NA	ND	1749
46 samples from exposed Americans prepared from over 900 specimens 80% lipid (Commoner et al., 1986)		11	NA	NA	34	NA	NA	NA	NA	22	22	NA	NA	75	1110
3 samples from Vietnam Veterans exposed to Agent Orange (average) (U.S. EPA, 1985b)		NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA

APPENDIX D
TABLE D.4

CDD AND CDF CONCENTRATIONS (ppt) IN HUMAN TISSUE

DESCRIPTION	2,3,7,8 Tetra-CDF	Tetra-CDF	1,2,3,7,8 Penta-CDF	2,3,4,7,8 Penta-CDF	Penta-CDF	1,2,3,4,7,8 Hexa-CDF	1,2,3,6,7,8 Hexa-CDF	2,3,4,6,7,8 Hexa-CDF	Hexa-CDF	1,2,3,4,6,7,8 Hepta-CDF	1,2,3,4,7,8,9 Hepta-CDF	Hepta-CDF	Octa-CDF	Total CDF
1 sample from deceased Yusho patient (Masuda and Yoshimura, 1984)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	A 6000-13000	
Liver Tissue														
1 deceased Yusho patient (Masuda and Yoshimura, 1984; Rappe et al., 1983b)	NA	<1	NA	10	NA	NA	NA	NA	55	100	NA	NA	<3	3000-25000
1 Yusho baby from Taiwan (Rappe et al., 1983b)	60	NA	194	91	NA	193	NA	NA	NA	NA	NA	NA	NA	NA
Blood														
Workers exposed to chlorophenol at a sawmill (Rappe et al., 1983b)	NA	NA	NA	NA	NA	NA	NA	NA	NA	40	NA	<1	<3	NA
1 sample taken in Seveso, Italy after accident (Facchetti et al., 1980)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA

APPENDIX D
TABLE D.4

CDD AND CDF CONCENTRATIONS (ppt) IN HUMAN TISSUE

DESCRIPTION	2,3,7,8 Tetra-CDF	Tetra-CDF	1,2,3,7,8 Penta-CDF	2,3,4,7,8 Penta-CDF	Penta-CDF	1,2,3,4,7,8 Hexa-CDF	1,2,3,6,7,8 Hexa-CDF	2,3,4,6,7,8 Hexa-CDF	Hexa-CDF	1,2,3,4,6,7,8 Hepta-CDF	1,2,3,4,7,8,9 Hepta-CDF	Hepta-CDF	Octa-CDF	Total CDF
Milk														
5 samples from South Vietnam in 1973 (average) (Commoner et al., 1986)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Milk Lipid														
5 samples from South Vietnam in 1973 (average) (Commoner et al., 1986)	NA	NA	NA	10	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA

NA = Not Available
ND = Not Detected
(Numbers in parentheses are limits of detection)
< = less than
* = reported as 1,2,3,4,7,8,9-heptaCDD

Analyses of the rice oil indicated that over 40 CDF congeners were present ranging from tri to hexaCDF. Table D.4 shows the concentrations of CDFs detected by Rappe et al. (1983b) and Masuda et al. (1984) in the adipose tissue and liver of a "Yusho" baby from Taiwan and in the adipose tissue of a deceased "Yusho" patient. In the liver sample the dominant congener was 1,2,3,7,8-pentaCDF, while in adipose tissue the highest value was found for 2,3,4,7,8-pentaCDF.

A recent U.S. EPA survey of 46 pooled tissue samples taken from 900 individuals believed to be representative of the general U.S. population showed CDDs and CDFs are generally present (Commoner et al., 1986). Comparable data for North and South Vietnamese samples are also reported. The levels in North Vietnam are about an order of magnitude below those found in either South Vietnam or the United States. It is believed by Commoner et al. that the concentrations of CDD and CDF in adipose tissue of South Vietnamese (higher than in the United States samples by a factor of 3.5) is indicative of the exposure to Agent Orange. The CDD and CDF concentrations in Americans are believed by the authors to probably originate from (1) CDD and CDF contaminated chemicals that enter the food chain from waste effluents or agricultural sprays, and (2) combustion of chlorine-containing fuels emitting particles that eventually enter the food chain or get inhaled directly.

ENVIRONMENTAL CONTAMINATION--CALIFORNIA

Monitoring for CDDs and CDFs has not been as extensive in California as it has been in other areas. Much of the data produced to date and presented in the previous section has been related to industrial, occupational, and waste disposal practices that have caused environmental contamination and/or human exposure. Most of the industrial production of herbicides, such as 2,4,5-T, and of chlorophenol products containing CDDs and CDFs as contaminants, has been in other states which has helped to minimize the occurrence of contamination in California. Waste products contaminated with CDDs and CDFs from the manufacturing of these products have also been less of a problem, although there are other chemical production processes (see Appendix B) which may produce them as byproducts, some of which are in use by the chemical industry in California.

CDDs and CDFs chlorinated in the 2,3,7, and 8 positions have recently been evaluated and recommended for classification as toxic air contaminants by the Air Resources Board in a joint effort with the Department of Health Services (CARB and CDHS, 1986). While no monitoring has been conducted to date, estimates of emission factors indicate that combustion sources such as solid waste incinerators may provide a significant contribution to the CDD and CDF input into the environment (CARB and CDHS,

1986; Wong, 1984). Anywhere from one to ten percent of the chlorophenols used in wood treatment operations may be burned with wood wastes (Bridle et al., 1984).

Hazardous waste site investigation and cleanup activities, which include wood treatment facilities, have provided evidence of CDD and CDF contamination in the State. The U.S. EPA National Dioxin study and a U.S. Fish and Wildlife study have demonstrated the presence of these toxicants in a limited number of fish and river sediment samples. The U.S. EPA indicates more monitoring in California for CDDs and CDFs related to the use of chlorophenols in the wood industry is likely in the future (U.S. EPA, 1986f).

Fish and Freshwater Sediment Studies

The U.S. Fish and Wildlife Service (1983) conducted a study in 1980 to 1981 to assess the effect of various water contaminants on striped bass populations. The study involved striped bass adults, young and eggs. While most samples were taken from rivers in the eastern U.S., the Sacramento River was included on the west coast, with analysis for all Cl_4 to Cl_8 CDDs and CDFs. The results, shown in Table D.5, indicate more CDFs than CDDs present in both whole fish and in fish eggs, with tetra- and pentaCDFs detected at higher levels than the higher chlorinated isomer groups. 2,3,7,8-TetraCDD was found only in the eggs, with 2,3,7,8-tetraCDF present in both whole fish and in the eggs.

As part of the National Dioxin Study by the U.S. EPA (1986e), samples were taken of fish and sediment from the Santa Ana River in southern California. This river was selected because it receives industrial and agricultural inputs from many sources. This study looked only for 2,3,7,8-tetraCDD, which was found in carp at a level of 4.6 ppt, and in a sediment sample from Mill Creek at a level of 0.6 ppt near its confluence with the Santa Ana River.

SUMMARY AND DISCUSSION

CDDs and CDFs are formed as contaminants from precursor compounds during the production of certain chemical products, and are released to the environment in the course of product use or disposal. Chemical products include, but are not limited to phenoxy herbicides, PCBs, diphenyl ether herbicides, and chlorophenol wood preservatives. CDD and CDF contaminated wastes generated in the manufacture and use of these products have also caused serious contamination through the use of unsound disposal practices.

Fish in several areas of the U.S. and other parts of the world have levels of CDDs and CDFs which have resulted in health advisories regarding consumption. Waste disposal practices and industrial process effluents are believed to be the sources of contamination.

TABLE D.5

CDD AND CDF CONCENTRATIONS IN CALIFORNIA FRESHWATER FISH AND SEDIMENT

Sample and Source	CDD (ppt) Tetra	Penta	Hexa	Hepta	Octa	CDF (ppt) Tetra	Penta	Hexa	Hepta	Octa
Carp Santa Ana River (U.S. EPA, 1986)	4.6[a]	-[b]	-	-	-	-	-	-	-	-
Sediment Mill Creek (U.S. EPA, 1986)	0.6[a]	-	-	-	-	-	-	-	-	-
Striped Bass Sacramento River (USFWS, 1983)	ND[c]	ND	ND	ND	ND	10	9	TR[d]	2	4.5
Striped Bass Eggs Sacramento River (USFWS, 1983)	6[a]	ND	ND	ND	ND	30	4	ND	ND	ND

a/ 2,3,7,8 Isomer
b/ Not Analyzed (-)
c/ Not Detected (ND)
d/ Trace (TR)

In Vietnam, the use of Agent Orange, a mixture of the phenoxy herbicides 2,4-D and 2,4,5-T containing CDDs and CDFs at low ppm levels as contaminants, has resulted in elevated levels of CDDs and CDFs in both humans and the environment. Similar studies of phenoxy herbicide use, especially 2,4,5-T in this country and in others, have determined product-related environmental and human residues.

Human exposure has resulted from several accidents, industrial and otherwise, which have caused some of the more significant contamination episodes. The evacuation of the town of Times Beach, Missouri due to the use of CDD and CDF contaminated oil for dust control on roads, the chemical explosion in Seveso, Italy in 1976 which caused large-scale contamination, and the ingestion of rice bran oil contaminated with CDFs (Yusho poisoning) in Japan and Taiwan are some of the more serious events affecting large numbers of people.

Several studies have determined the presence of CDDs and CDFs in tissues from the general population, indicating a probable steady background exposure. These compounds have also been found in places far from discrete sources. Combustion of CDD and CDF containing wastes and wastes which contain precursor materials capable of forming them, is believed to be a significant source and may be responsible for their ubiquitous presence in the environment. The California Air Resources Board has recently recommended that CDDs and CDFs be classified as toxic air contaminants, based in part on an evaluation of adverse health effects conducted by the California Department of Health Services.

In California, CDDs and CDFs have been determined as contaminants during investigations of hazardous waste sites, many of which are locations of present and former wood treatment operations. Levels have also been found in limited fish and river sediment samples in the State.

APPENDIX H

RESULTS OF STATE BOARD CDD AND CDF

2,3,7,8 CONGENER-SPECIFIC ANALYSES

TABLE H.1

RESULTS OF CDD AND CDF 2,3,7,8 CONGENER SPECIFIC ANALYSIS, PHASE 1: SAWMILL A: COMPARISON OF ON-SITE DRUM OF COMMERCIAL PENTACHLOROPHENATE FORMULATION AND SLUDGE FROM DIP TANK

	COMMERCIAL Na-PCP		DIP TANK SLUDGE	
	CAL[1/]	SWEDEN[2/]	CAL[1/]	SWEDEN[2/]
CDDs (ppb)				
Total TetraCDD	1.9	<1	DL 2.2	<1
2,3,7,8	DL 1.4	<1	DL 2.7	<1
% 2,3,7,8 of total tetraCDD	-	-	-	-
Total PentaCDD	140	304	28	107
1,2,3,7,8	28.3	24	DL 15.9	10
% 2,3,7,8 of total pentaCDD	20%	13%	-	9%
Total HexaCDD	14,000	5,700	3,630	2,600
1,2,3,4,7,8	DL 6.1	ND	DL 12.5	ND
1,2,3,6,7,8	4,050	3,600	1,790	1,800
1,2,3,7,8,9	ND	ND	NR	ND
Total 2,3,7,8	4,050	3,600	1,790	1,800
% 2,3,7,8 of total hexaCDD	29%	63%	49%	69%
Total Hepta-CDD	100,000	40,000	36,400	42,000
1,2,3,4,6,7,8	33,800	28,000	15,400	25,000
% 2,3,7,8 of total heptaCDD	34%	70%	42%	60%
OctaCDD	81,000	13,000	115,000	155,000

FOOTNOTES

1/ Isomer group totals quantified on DB-5 column; specific 2,3,7,8 congeners quantified on Supelco 2331 column; NR = not reported; DL 1.4 = not detected at detection limit of 1.4 ppb.

2/ ND is less than 50 ppb; <1 = reported as less than 1 ppb.

TABLE H.1

RESULTS OF CDD AND CDF 2,3,7,8 CONGENER SPECIFIC ANALYSIS, PHASE 1: SAWMILL A: COMPARISON OF ON-SITE DRUM OF COMMERCIAL PENTACHLOROPHENATE FORMULATION AND SLUDGE FROM DIP TANK

	COMMERCIAL Na-PCP		DIP TANK SLUDGE	
	CAL[1/]	SWEDEN[2/]	CAL[1/]	SWEDEN[2/]
CDDs (ppb)				
Total TetraCDD	1.9	<1	DL 2.2	<1
2,3,7,8	DL 1.4	<1	DL 2.7	<1
% 2,3,7,8 of total tetraCDD	-	-	-	-
Total PentaCDD	140	304	28	107
1,2,3,7,8	28.3	24	DL 15.9	10
% 2,3,7,8 of total pentaCDD	20%	13%	-	9%
Total HexaCDD	14,000	5,700	3,630	2,600
1,2,3,4,7,8	DL 6.1	ND	DL 12.5	ND
1,2,3,6,7,8	4,050	3,600	1,790	1,800
1,2,3,7,8,9	ND	ND	NR	ND
Total 2,3,7,8	4,050	3,600	1,790	1,800
% 2,3,7,8 of total hexaCDD	29%	63%	49%	69%
Total Hepta-CDD	100,000	40,000	36,400	42,000
1,2,3,4,6,7,8	33,800	28,000	15,400	25,000
% 2,3,7,8 of total heptaCDD	34%	70%	42%	60%
OctaCDD	81,000	13,000	115,000	155,000

FOOTNOTES

1/ Isomer group totals quantified on DB-5 column; specific 2,3,7,8 congeners quantified on Supelco 2331 column; NR = not reported; DL 1.4 = not detected at detection limit of 1.4 ppb.

2/ ND is less than 50 ppb; <1 = reported as less than 1 ppb.

TABLE H.1 (continued)

RESULTS OF CDD AND CDF 2,3,7,8 CONGENER SPECIFIC ANALYSIS, PHASE 1: SAWMILL A: COMPARISON OF ON-SITE DRUM OF COMMERCIAL PENTACHLOROPHENATE FORMULATION AND SLUDGE FROM DIP TANK

	COMMERCIAL Na-PCP		DIP TANK SLUDGE	
	CAL[1/]	SWEDEN[2/]	CAL[1/]	SWEDEN[2/]
CDFs (ppb)				
Total TetraCDF	1,200	1,671	412	354
2,3,7,8	149	253	140	69
% 2,3,7,8 of total tetraCDF	12%	15%	34%	19%
Total PentaCDF	6,400	10,000	2,970	5,100
1,2,3,7,8	319	265	131	80
2,3,4,7,8	324	319	119	110
Total 2,3,7,8	643	584	250	190
% 2,3,7,8 of total pentaCDF	10%	6%	8%	4%
Total HexaCDF	49,000	7,200	8,700	8,500
1,2,3,4,7,8	DL 2.8	200	DL 8.5	300
1,2,3,6,7,8	225		145	
1,2,3,7,8,9	480	300	DL 14.3	300
2,3,4,6,7,8	DL 385	<100	DL 16.5	<100
Total 2,3,7,8	705	500	145	600
% 2,3,7,8 of total hexaCDF	1.4%	7%	2%	7%
Total HeptaCDF	91,000	9,700	9,300	8,000
1,2,3,4,6,7,8	6,190	3,900	2,270	3,000
1,2,3,4,7,8,9	154	ND	DL 100	<100
Total 2,3,7,8	6,344	3,900	2,270	3,000
% 2,3,7,8 of total heptaCDF	7%	40%	24%	38%
OctaCDF	36,000	1,000	3,890	1,800

FOOTNOTES

1/ Isomer group totals quantified on DB-5 column; specific 2,3,7,8 congeners quantified on Supelco 2331 column; NR = not reported; DL 1.0 = not detected at detection limit of 1.0 ppb.

2/ Sweden: 1,2,3,7,8- and 1,2,3,4,8-PentaCDF co-elute; 1,2,3,4,7,9-, 1,2,3,4,7,8- and 1,2,3,6,7,8-HexaCDF co-elute; ND is less than 50 ppb; <100 = reported as less than 100 ppb.

TABLE H.2

RESULTS OF CDD AND CDF CONGENER SPECIFIC ANALYSIS,
PHASE 1: SAWMILL B: COMPARISON OF WET AND DRY SLUDGES
SAMPLED FROM ABANDONED DIP TANK

	WET SLUDGE		DRY SLUDGE	
	CAL[1/]	SWEDEN[2/]	CAL[1/]	SWEDEN[2/]
CDDs (ppb)				
Total TetraCDD	DL 1.8	<1	8.4	60
2,3,7,8	DL 2.1	<1	8.3	11
% 2,3,7,8 of total tetraCDD	-	-	99%	18%
Total PentaCDD	25	246	720	1,298
1,2,3,7,8	34.3	14	185	212
% 2,3,7,8 of total pentaCDD	64%[3/]	5.7%	26%	16%
Total HexaCDD	640	400	6,200	8,600
1,2,3,4,7,8	DL 3.5	ND	93	ND
1,2,3,6,7,8	316	200	2,980	5,300
1,2,3,7,8,9	NR	ND	NR	ND
Total 2,3,7,8	316	200	3,073	5,300
% 2,3,7,8 of total hexaCDD	49%	50%	50%	62%
Total HeptaCDD	3,000	3,400	14,000	22,000
1,2,3,4,6,7,8	1,600	2,200	8,030	14,000
% 2,3,7,8 of total heptaCDD	53%	65%	57%	64%
OctaCDD	7,200	1,600	63,000	40,000

FOOTNOTES

1/ Isomer group totals quantified on DB-5 column; specific 2,3,7,8 congeners quantified on Supelco 2331 column; NR = not reported; DL 1.4 = not detected at detection limit of 1.4 ppb.

2/ ND is less than 50 ppb; <1 = reported as less than 1 ppb.

3/ 64% based on DB-5 column; a previous analysis reported 19% of the total as 1,2,3,7,8-pentaCDD.

TABLE H.2 (continued)

RESULTS OF CDD AND CDF 2,3,7,8 CONGENER SPECIFIC ANALYSIS,
PHASE 1: SAWMILL B: COMPARISON OF WET AND DRY SLUDGES
SAMPLED FROM ABANDONED DIP TANK

	WET SLUDGE		DRY SLUDGE	
	CAL[1/]	SWEDEN[2/]	CAL[1/]	SWEDEN[2/]
CDFs (ppb)				
Total TetraCDF	76	79	1,700	2,294
2,3,7,8	20	13	112	78
% 2,3,7,8 of total tetraCDF	26%	16%	7%	3%
Total PentaCDF	650	500	9,100	13,000
1,2,3,7,8	24	25	140	119
2,3,4,7,8	19	31	131	169
Total 2,3,7,8	43	56	271	288
% 2,3,7,8 of total pentaCDF	7%	11%	3%	2%
Total HexaCDF	1,530	900	6,600	9,200
1,2,3,4,7,8	DL 3.9	ND	DL 14	100
1,2,3,6,7,8	19		104	
1,2,3,7,8,9	DL 11.3	ND	DL 23.5	200
2,3,4,6,7,8	26	ND	177	ND
Total 2,3,7,8	45	-	281	300
% 2,3,7,8 of total hexaCDF	3%	0	4%	3%
Total HeptaCDF	960	700	3,000	4,100
1,2,3,4,6,7,8	446	300	1,120	1,900
1,2,3,4,7,8,9	DL 17	ND	DL 32.5	ND
Total 2,3,7,8	446	300	1,120	1,900
% 2,3,7,8 of total heptaCDF	46%	43%	37%	46%
OctaCDF	270	100	2,400	600

FOOTNOTES

1/ Isomer group totals quantified on DB-5 column; specific 2,3,7,8 congeners quantified on Supelco 2331 column; NR = not reported; DL 1.4 = not detected at detection limit of 1.4 ppb.

2/ Sweden: 1,2,3,7,8- and 1,2,3,4,8-PentaCDF co-elute; 1,2,3,4,7,9-, 1,2,3,4,7,8- and 1,2,3,6,7,8-HexaCDF co-elute; ND is less than 50 ppb; <100 = reported as less than 100 ppb.

TABLE H.3

RESULTS OF CDD AND CDF 2,3,7,8 CONGENER SPECIFIC ANALYSIS,
PHASE 2: SAWMILL C: TETRACHLOROPHENATE

	TETRA-CHLOROPHENATE DRUM		DIP TANK LIQUID		DIP TANK SLUDGE		DIP TANK SLUDGE	
	CAL[1]	IITRI[2]	CAL[1]	IITRI[2]	CAL[1]	IITRI[2]	CAL[1]	IITRI[2]
CDDS (ppb)								
Total TetraCDD	4.4	<1.7[3]	0.67	0.2	3.3	<3.0	8.3	6.5
2,3,7,8	<2.1	<1.7	<.10	<0.03	<1.5	<3.0	6.8	<1.7
% 2,3,7,8 of total tetraCDD	-	-	-	-	-	-	82%	-
Total PentaCDD	256	21	1.4	0.5	39.7	20	40.8	31
1,2,3,7,8	<5.1	<2.0	<.12	<0.03	<5.3	<1.7	<4.3	<0.9
% 2,3,7,8 of total pentaCDD	-	-	-	-	-	-	-	-
Total HexaCDD	1,630	851	15.5	7.6	410	397	532	421
1,2,3,4,7,8	<12.2	<4.7	8.2	<0.04	218	<3.0	<10.1	<2.6
1,2,3,6,7,8	667	323		3.3		192	242	192
1,2,3,7,8,9	NA[4]	14	NA	0.2	NA	7.9	NA	7
total 2,3,7,8	667	337	8.2	3.5	218	200	242	199
% 2,3,7,8 of total hexaCDD	41%	40%	53%	46%	53%	50%	45%	47%
Total HeptaCDD	1,360	805	25.7	14	1,380	1,564	1,720	1,444
1,2,3,4,6,7,8	849	527	15.3	9.3	797	974	976	898
% 2,3,7,8 of total heptaCDD	62%	65%	60%	66%	58%	62%	57%	62%
OctaCDD	1,450	5,680	106	289	1,290	9,770	4,960	11,056

FOOTNOTES

1/ Cal Labs - totals quantified on DB-5 column; Congener specific quantified on SP 2331 column.

2/ IITRI - quantified on SP 2330.

3/ <1.0 indicates not detected at a detection limit of 1.0 ppb.

4/ NA - Not analyzed.

TABLE H.3 (continued)

RESULTS OF CDD AND CDF 2,3,7,8 CONGENER SPECIFIC ANALYSIS,
PHASE 2: SAWMILL C: TETRACHLOROPHENATE

	TETRACHLOROPHENATE DRUM		DIP TANK LIQUID		DIP TANK SLUDGE		DIP TANK SLUDGE	
	CAL[1]	IITRI[2]	CAL[1]	IITRI[2]	CAL[1]	IITRI[2]	CAL[1]	IITRI[2]
CDFs (ppb)								
Total TetraCDF	1,280	1,179	10.5	5.3	234	534	279	522
2,3,7,8	262	138	2.8	1.2	46	62	65	64
% 2,3,7,8 of total tetraCD	20%	12%	27%	23%	20%	12%	23%	12%
Total PentaCDF	7,190	1,765	53	13	1,150	658	1,310	555
1,2,3,7,8 & 1,2,3,4,8	262	<2.0[3]	2.1	1.5	50	95	54	82
2,3,4,7,8	88	59	1.3	0.6	28	60	26	50
total 2,3,7,8	350	59	3.4	2.1	77	155	80	132
% 2,3,7,8 of total pentaCDF	5%	3%	6%	16%	7%	24%	6%	24%
Total HexaCDF	6,240	4,657	58	36	1,470	1,806	1,850	2,227
1,2,3,4,7,8 & 1,2,3,4,7,9	20	<4.7	0.09	0.2	16	18	17	23
1,2,3,6,7,8	<12.5	<4.7	22	0.1		7.6	6	9.2
1,2,3,7,8,9	46	<4.7	<.23	<0.04	<11.1	<3.0	31	<2.6
2,3,4,6,7,8	<14.6	30	0.94	0.4	<7.0	28	<7.5	36
total 2,3,7,8	66	30	23	0.7	16	54	54	68
% 2,3,7,8 of total hexaCDF	1%	0.6%	40%	2%	1%	3%	3%	3%
Total HeptaCDF	2,750	1,338	32	14	963	921	1,180	774
1,2,3,4,6,7,8	808	582	11	6.3	306	402	342	341
1,2,3,4,7,8,9	<18.7	<3.7	<.36	<0.03	<10.6	519	<7.1	<1.9
total 2,3,7,8	808	582	11	6.3	306	921	342	341
%2,3,7,8 of total heptaCDF	29%	43%	34%	45%	32%	100%	29%	44%
OctaCDF	132	<337	6.3	1.1	378	102	375	100

1/ Cal Labs - totals quantified on DB-5 column; Congener specific quantified on SP 2331 column.

2/ IITRI - SP 2330 column - note co-eluting pentaCDF and hexaCDF congener.

3/ <1.0 indicates not detected at a detection limit of 1.0 ppb.

TABLE H.4

RESULTS OF CDD AND CDF 2,3,7,8 CONGENER SPECIFIC ANALYSIS,
PHASE 2: WOOD TREATMENT PLANT

CDDs (ppb)	"BLOOM" CAL[1]	"BLOOM" IITRI[2]	COMMERCIAL" CAL[1]	COMMERCIAL" IITRI[2]	SOIL (MOUTH OF RETORT) CAL[1]	SOIL (MOUTH OF RETORT) IITRI[2]	SUMP (LIQUID) CAL[1]	SUMP (LIQUID) IITRI[2]
Total TetraCDD	<2.0[3]	<1.8	<.32	<2.3	<.48	<1.1	<1.4	5.5
2,3,7,8	<3.0	<1.8	<2.7	<2.3	<2.2	<1.1	<4.7	<0.7
% 2,3,7,8 of total tetraCDD	-	-	-	-	-	-	-	-
Total PentaCDD	109	30	<1.3	<3.5	4.4	<1.2	39	<1.4
	90	21	<14.3	<3.5	<12.8	<1.2	<19.7	<1.4
1,2,3,7,8								
% 2,3,7,8 of total pentaCDD	82%	70%	-	-	-	-	-	-
Total HexaCDD	2,020	1,140	127	144	215	271	1,420	84
1,2,3,4,7,8	145	103	<8.4	<16.6	<12.3	9.4	30	12
1,2,3,6,7,8	510	347	65	<16.6	70	90	384	28
1,2,3,7,8,9	NA[4]	8	NA[4]	<16.6	NA[4]	22	NA[4]	14
TOTAL 2,3,7,8	655	458	65	-	70	121	414	54
% 2,3,7,8 of total HexaCDD	32%	40%	51%	-	33%	45%	29%	64%
Total HeptaCDD	31,800	21,552	8,490	10,568	1,940	2,890	12,900	548
1,2,3,4,6,7,8	30,300	16,837	5,980	7,319	1,204	1,839	8,270	343
% 2,3,7,8 of total HeptaCDD	95%	78%	70%	69%	62%	64%	64%	63%
OctaCDD	135,000	145,693	115,000	143,043	8,040	8,397	77,090	10,707

FOOTNOTES

1/ Cal Labs - totals quantified on DB-5 column; Congener specific quantified on SP 2331.

2/ IITRI - quantified on SP 2330.

3/ <1.0 indicates not detected at a detection limit of 1.0 ppb.

4/ NA - Not analyzed.

TABLE H.4 (continued)

RESULTS OF CDD AND CDF 2,3,7,8 CONGENER SPECIFIC ANALYSIS,
PHASE 2: WOOD TREATMENT PLANT

	"BLOOM"		COMMERCIAL"		SOIL (MOUTH OF RETORT)		SUMP (LIQUID)	
CDFs (ppb)	CAL[1]	IITRI[2]	CAL[1]	IITRI[2]	CAL[1]	IITRI[2]	CAL[1]	IITRI[2]
Total TetraCDF	18	43	<0.2[3]	1.3	0.67	4.9	9.1	26
2,3,7,8	4.4	4.4	<3.6	<1.9	<4.7	<1.3	<5.2	<1.3
% 2,3,7,8 of total tetraCDF	24%	10%	-	-	-	-	-	-
Total PentaCDF	561	203	<2.9	<3.5	72.6	12	484	27
1,2,3,7,8 &								
1,2,3,4,8	53	<2.2	<9.1	<3.5	<7.2	4.8	32	8.4
2,3,4,7,8	31	27	<9.1	<3.5	<5.5	<1.2	18	4.2
total 2,3,7,8	84	27	-	-	-	4.8	50	13
% 2,3,7,8 of total pentaCDF	15%	13%	-	-	-	40%	10%	47%
Total HexaCDF	4,520	3,637	251	209	211	219	2,440	168
1,2,3,4,7,8 &								
1,2,3,4,7,9	902	662	12.8	<16.6	6.0	<5.0	61	<3.6
1,2,3,6,7,8	236	232	<12.1	<16.6	<11.8	<5.0	25	<3.6
1,2,3,7,8,9	94	<3.9	<14.4	<16.6	<17.8	<5.0	50	<3.6
2,3,4,6,7,8	<8.7	85	<14.3	37	<13.4	17	<20.6	<3.6
total 2,3,7,8	1,232	979	13	37	6.0	17	136	-
% 2,3,7,8 of total hexaCDF	27%	27%	5%	18%	3%	8%	6%	-
Total HeptaCDF	17,400	8,606	4,240	2,613	458	388	2,590	111
1,2,3,4,6,7,8	11,600	5,056	747	425	747	145	900	40
1,2,3,4,7,8,9	1,490	396	110	120	110	12	<50	16
total 2,3,7,8	13,090	5,452	857	545	847	157	900	56
%2,3,7,8 of total heptaCDF	75%	63%	20%	21%	185%	40%	35%	50%
OctaCDF	223,000	12,323	175,000	11,648	1,470	222	3,600	<326

FOOTNOTES

1/ Cal Labs - totals quantified on DB-5 column; Congener specific quantified on SP 2331 column.

2/ IITRI - quantified on SP 2330.

3/ <1.0 indicates not detected at a detection limit of 1.0 ppb.

TABLE H.5

CDD AND CDF 2,3,7,8 CONGENER SPECIFIC ANALYSES:
PERCENT SPECIFIC 2,3,7,8 ISOMERS DETECTED IN EACH
CDD AND CDF ISOMER GROUP. PHASE 1: SAWMILLS A AND B

	COMMERCIAL Na-PCP FORMULATION	SAWMILL A SLUDGE	SAWMILL B LIQUID SLUDGE	SAWMILL B DRY SLUDGE
CDDs				
2,3,7,8 tetraCDD[1]	ND	ND	ND	18% 99%[3]
1,2,3,7,8 pentaCDD[2]	12%	ND 9%[3]	5.7% 38%[3]	20%
1,2,3,4,7,8 hexaCDD[2]	ND	ND	ND	ND 1.5%[3]
1,2,3,6,7,8 hexaCDD	39%	58%	50%	56%
1,2,3,7,8,9 hexaCDD	ND	ND	ND	ND
1,2,3,4,6,7,8 heptaCDD[2]	44%	60%	64%	61%
CDFs				
2,3,7,8 tetraCDF[1]	14%	27%	22%	4.8%
1,2,3,7,8 pentaCDF	3.6%	2.6%	4.3%	1.2%
2,3,4,7,8 pentaCDF	3.9%	2.8%	4.3%	1.4%
1,2,3,4,7,8 hexaCDF	ND	ND	ND	ND
1,2,3,6,7,8 hexaCDF	0.8%	2.6%	ND 1.2%[3]	1.3%
1,2,3,7,8,9 hexaCDF	1.3%	ND 3.5%[3]	ND	ND 2.2%[3]
2,3,4,6,7,8 hexaCDF	ND	ND	ND 1.7%[3]	ND 2.7%[3]
1,2,3,4,6,7,8 heptaCDF	10%	30%	45%	43%
1,2,3,4,7,8,9 heptaCDF	<0.2%	ND	ND	ND

FOOTNOTES

ND = None Detected
1/ Internal standard used by both labs (also used OctaCDD).
2/ Internal standard used by Cal Labs.
3/ Results of both labs given.

TABLE H.5 (continued)

CDD AND CDF 2,3,7,8 CONGENER SPECIFIC ANALYSES: PERCENT SPECIFIC 2,3,7,8 ISOMERS DETECTED IN EACH ISOMER GROUP. PHASE 2: SAWMILL C, TETRACHLOROPHENATE

	COMMERCIAL TETRACHLORO-PHENATE FORMULATION	DIP TANK LIQUID	DIP TANK SLUDGE	DIP TANK SLUDGE
CDDs				
2,3,7,8 tetraCDD[1]	ND	ND	ND	82% ND[4]
1,2,3,7,8 pentaCDD[2]	ND	ND	ND	ND
1,2,3,4,7,8 hexaCDD[2]	ND	ND	ND	ND
1,2,3,6,7,8 hexaCDD	40%	50%	51%	54%
1,2,3,7,8,9 hexaCDD	NA 1.6%[4]	NA 2.6%[4]	NA[4] 2.0%	NA[4] 1.7%
1,2,3,4,6,7,8 heptaCDD[2]	64%	60%	60%	59%
CDFs				
2,3,7,8 tetraCDF[1]	16%	24%	14%	16%
1,2,3,7,8 pentaCDF[3]	3.6% ND[4]	5.5%	8%	8.4%
2,3,4,7,8 pentaCDF	1.6%	2.9%	4.7%	4.1%
1,2,3,4,7,8 hexaCDF[3]	0.3% ND[4]	3.1%	1%	1%
1,2,3,6,7,8 hexaCDF	ND	24%	0.4%	0.7%
1,2,3,7,8,9 hexaCDF	0.7% ND[4]	ND	ND	1.7% ND[4]
2,3,4,6,7,8 hexaCDF	ND 0.6%[4]	1.4%	ND 1.6%	ND[4] 1.6%[4]
1,2,3,4,6,7,8 heptaCDF	34%	38%	38%	35%
1,2,3,4,7,8,9 heptaCDF	ND	ND	ND 56%[4]	ND

FOOTNOTES

ND = None Detected
NA = Not Analyzed

1/ Internal standard used by both labs (also used OctaCDD).
2/ Internal standard used by Cal Labs.
3/ Internal standard used by IITRI.
4/ Results of both labs given; first value is Cal Labs, second is IITRI.

TABLE H.5 (continued)

CDD AND CDF 2,3,7,8 CONGENER SPECIFIC ANALYSES:
PERCENT SPECIFIC 2,3,7,8 ISOMERS DETECTED IN EACH
ISOMER GROUP. PHASE 2: WOOD TREATMENT PLANT: COMMERCIAL PCP

	"BLOOM"	"COMMERCIAL"	SOIL AT RETORT MOUTH	SUMP LIQUID
CDDs				
2,3,7,8 tetraCDD[1]	ND	ND	ND	ND
1,2,3,7,8 pentaCDD[2]	80%	ND	ND	ND
1,2,3,4,7,8 hexaCDD[2]	7.8%	ND	ND 3.5% [4]	2.1% 14%[4]
1,2,3,6,7,8 hexaCDD	27%	51%	33%	27% 33%[4]
1,2,3,7,8,9 hexaCDD	NA 0.7%[4]	NA ND[4]	NA 8.1%[4]	NA 17%[4]
1,2,3,4,6,7,8 heptaCDD	88%	70%	63%	64%
CDFs				
2,3,7,8 tetraCDF[1]	14%	ND	ND	ND
1,2,3,7,8 pentaCDF[3]	9.4% ND[4]	ND	ND 40%[4]	6.6% 31%[4]
2,3,4,7,8 pentaCDF	5.5% 13%[4]	ND	ND	3.7% 16%[4]
1,2,3,4,7,8 hexaCDF[3]	19%	5.1% ND[4]	2.8% ND[4]	2.5% ND[4]
1,2,3,6,7,8 hexaCDF	5.7%	ND	ND	1% ND[4]
1,2,3,7,8,9 hexaCDF	2.1% ND[4]	ND	ND	2% ND[4]
2,3,4,6,7,8 hexaCDF	ND 2.3%[4]	ND 18%[4]	ND 7.8%[4]	ND
1,2,3,4,6,7,8 heptaCDF	64%	17%	163% 37%[4]	35%
1,2,3,4,7,8,9 heptaCDF	7.3%	3.1%	24% 3.1%[4]	ND 14%[4]

FOOTNOTES

ND = None Detected
NA = Not Analyzed

1/ Internal standard used by both labs (also used OctaCDD).
2/ Internal standard used by Cal Labs.
3/ Internal standard used by IITRI.
4/ Results of both labs given; first value is Cal Labs, second is IITRI.

APPENDIX J

BACKGROUND DOCUMENTS FOR CHAPTER 8: HAZARD EVALUATION

PAGE

BACKGROUND DOCUMENTS:

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CALIFORNIA SITE MITIGATION DECISION TREE MANUAL

EXECUTIVE SUMMARY

Introduction

The purpose of the California Site Mitigation Decision Tree is to provide State decision makers with a standardized approach to setting site-specific mitigation criteria. The Decision Tree will also facilitate evaluation of remedial action alternatives to select the best plan based on scientific considerations of factors relating to public health and the environment while considering demographic factors, local concerns, and other variables.

Major elements contained within the Decision Tree include processes for setting Applied Action Levels (AALs) for contaminants in soil, water, air and biota in an expeditious manner; identifying specific data to be gathered; identifying preferred data gathering techniques and developing site mitigation criteria for alternative remedial actions.

It should be noted that the Decision Tree process establishes both Applied Action Levels (AALs) and site mitigation criteria. AALs are exposure criteria applied to all sites throughout the State. AALs delineate concentrations of toxic substances that, when exceeded, place a specified biological receptor at significant risk. Because AALs are biological receptor specific, not site specific, they have statewide applicability. The mitigation criteria, on the other hand, are site-specific criteria that a remedial action must achieve to keep the exposure level at the biological receptor below the AAL (i.e., below significant risk).

The Decision Tree process consists of five components. These components include Preliminary Site Appraisal, Site Assessment, Risk Appraisal, Environmental Fate and Risk Determination, and Determination of Mitigation Strategy and Remedial Action Plan Selection. The relationship between these components and an overview of the Decision Tree process are shown in Figure 1.

The Decision Tree process was designed to be applied to a variety of sites that may range from small, relatively simple sites to large, highly complex and difficult sites. Because of this diversity, the Decision Tree document is rather massive. However, its size should not intimidate the user. If the site is small and relatively easy to mitigate, many of the decision branches are never opened and the decision process is very rapid. If the site is complex, many of the decision branches must be opened and pursued. In either case, the decision points and the data requirements needed to make sound decisions are defined.

COMPONENT I: Preliminary Site Appraisal

Sites in California contaminated with hazardous wastes have been identified by regulatory agencies such as the State of California Department of Health Services, the California State Water Resources Control Board and the nine Regional Water Quality Control Boards, the United States Environmental Protection Agency, and a myriad of local agencies responsible property owners reporting contamination problems; and concerned citizens, including residents and past and present employees of companies that have contaminated sites. Preliminary Site Appraisal (PSA) is initiated by the discovery of a site which is potentially contaminated with hazardous substances. Based on the

characteristics of the wastes present and the features of the site itself, the site may be determined to be sufficiently hazardous to be placed on either the National Priority List (NPL) and/or the State Bond Act Expenditure Plan (CSBAEP).

The process to rank sites on the NPL and CSBAEP is primarily based on qualitative information, and does not include the detailed investigation required to fully characterize a site. Therefore, additional information may be required to implement subsequent components of the Decision Tree process. The Decision Tree process may also be used for sites that are not listed on the NPL and CSBAEP.

The PSA component of the Decision Tree identifies the universe of contaminants potentially present at the site using information acquired from the NPL or CSBAEP process. Additional investigation may be needed regarding the use of chemicals, production of wastes, and disposal practices at the site. The universe of contaminants is verified by a limited sampling program of the areas where contamination is most likely to be found.

An initial investigation of the adverse effects of the contaminants is necessary to determine the potential endangerment to human health. Determination of the likelihood of adverse effects occurring upon exposure to the contaminants is based upon available toxicologic data. Available data may show that adverse human health effects are imminent, and appropriate emergency action is necessary to protect public health.

Determination of the occurrence of health effects due to low level, chronic exposure is more difficult and requires additional investigation of the extent and magnitude of contamination, and the toxicologic properties of the contaminants, individually and collectively. Once it has been established that additional site assessment is necessary to provide data to develop appropriate remedial measures, the remaining components of the Decision Tree are initiated.

COMPONENT II: Site Assessment

The second component of the Decision Tree includes a preliminary evaluation of the site specific factors that affect the tendency of a hazardous substance to move between environmental compartments (air, soil, water, and biota). First, critical exposure pathways are identified. These pathways are the means by which exposure to contaminants in air, soil, water and biota occurs. Water pathways include both surface and ground waters.

In the first phase of identifying critical exposure pathways, determination of the current contaminant concentrations at points of exposure to biological receptors of concern should be made. The measurements or estimates made at this point are not thought of as static; concentrations change with time. Hazardous substances are not assumed to be isolated from biological receptors in separate environmental compartments. The Decision Tree process identifies data to be gathered for each pathway and cites preferred methods of data collection.

Typically, in this initial stage, samples are taken of waste, surface soils and shallow soils, runoff and surface water and ground water from existing wells. Sampling data from these site assessment activities will be the basis for a decision as to whether expanded air, soils, and ground water investigations will be necessary.

Expanded Assessment

If an expanded assessment of the site is warranted, evaluation of the environmental setting as it affects the behavior of the contaminants is required. The quantification of the meteorological, biological, soils and hydrologic systems at the site, together with information about the chemical and physical properties of the contaminants, forms a basis to evaluate the environmental fate of contaminants.

The collection of the necessary and sufficient data to adequately characterize the site and contaminants is the principal objective of this component. Site investigation programs are iterative in nature and will often require subsequent sampling and monitoring device installation to resolve issues that arise after data collected during initial site appraisal are analyzed. Although environmental compartments are presented as separate modules, contaminant transfer occurs across compartment boundaries and processes in one compartment often influence processes in another.

Quality Assurance/Quality Control

As the process is described in the Decision Tree Manual, data obtained from field sampling and analysis and/or literature values may be used to determine existing and future concentrations of chemical contaminants in environmental media (i.e., air, soil, water, and biota). Regardless of the source, it is important that these data be accurate, precise, complete, representative and comparable to other appropriate data.

To ensure that all data used in the process described in this manual are representative of environmental conditions, the Quality Assurance/Quality Control Plans (QA/QC) used in the data generation need to be evaluated. The main components of a QA/QC plan that need special scrutiny are the basis for measurement, experimental information, statistical information (e.g., means, ranges, and standard deviation), and corroborative information.

The QA/QC plans for sampling and for analysis should be developed together. At a minimum, the data generators and users should work together in developing an integrated site-specific QA/QC plan.

COMPONENT III: Risk Appraisal

An evaluation of the effects produced by toxic substances which originate from waste sites centers on appraising the adverse impacts of these substances on the public health and the surrounding ecosystem. Every potential effect is not, and should not be, delineated by the appraisal process. Given the limited resources that are available and the complexity of the numerous sites scattered throughout California, the appraisal must focus on those biological receptors of concern that are potentially at risk. The Decision Tree process is aimed at ensuring their protection.

Three types of information are essential to evaluate the sites.

1. The toxic substances are identified from data collected in the Site Assessment Process.

2. The biological receptors of concern in the ecosystem potentially impacted by the toxic substances are identified in the Site Assessment Process.

3. The critical exposure pathways are delineated in the Site Assessment Process.

A criterion will be identified or developed for maximum acceptable exposure for toxic contaminants. The criteria are employed to identify significant adverse effects of the toxic contaminants on the biological receptors. These criteria, denoted as Applied Action Levels, are applicable statewide. An Applied Action Level (AAL) is specific to a toxic substance, a biological receptor and a medium of exposure.

The methodology employed to develop AALs is quite conventional. It is a compilation of the approaches outlined by the U.S. Environmental Protection Agency, the National Academy of Sciences and the California Department of Health Services. Toxic Substances are grouped into two catagories for the purpose of developing AALs. For carcinogens, mutagens and genotoxic teratogens no threshold for an adverse effect is assumed. The AALs are based on a maximum exposure level (MEL) which produces one adverse effect in a population of one million exposed. The MELs are determined from epidemiological research or long-term animal bioassays.

For other toxic agents a threshold for an adverse effect is assumed. The AAL is established, with a margin of safety, at the maximum exposure level which does not produce an adverse effect. Uncertainties associated with the above

approaches are factored into the criteria to ensure a margin of protection for the biological receptors of concern.

The total exposure of biological receptors to toxic substances via various media is evaluated at their site(s) of exposure. The fractions of the AAL present in each media of exposure are added. When the total cumulative exposure exceeds an MEL, a significant risk to a biological receptor is indicated and a risk management process is warranted.

Exposure to substances that produce the same toxic manifestation or are considered likely to interact is also appraised. When the total cumulative exposures to toxic chemicals in all media of exposure constitute a significant risk to a biological receptor, the initiation of a risk management process is warranted.

Whenever an Applied Action Level is exceeded in any media of exposure, an assessment will be made to determine the necessity for interim actions to be implemented immediately to protect public health and the environment. A few examples of immediate interim actions are: fencing the contaminated site, covering exposed contaminated soils, and restricting use of water. Immediate interim actions will usually not be the ultimate containment or treatment strategy. Interim actions are developed primarily to reduce public exposure prior to initiating the final Remedial Action Plan (RAP).

COMPONENT IV: Environmental Fate and Risk Determination

Environmental Fate - Subsurface Conditions, Soils and Groundwater

In producing this section of the Decision Tree Manual, it is recognized that diverse subsurface conditions are encountered in hazardous waste site investigations and that considerable flexibility and professional judgement are often required to conduct an investigation of subsurface geology, hydrology, and soil and ground water contamination. Items to be considered include those factors that could act to transfer contaminants adsorbed to soil particles through the soil column. Factors of concern are: infiltration of precipitation, leakage of liquids from underground storage or conveyance structures, and spillage or other discharges to ground that could encourage leaching of contaminants from soil. In many cases, both current and future land use must be considered to evaluate the effect of environmental factors on the contaminants residing in the soil column.

Patterns of soil contamination existing at a hazardous waste site may often be the result of waste disposal events that took place over many years. Variations in the waste type, climate and precipitation, micro-structure of the soils, biological activity, and soil-chemical interactions can act to result in a complex pattern of soil contamination. Evaluation of patterns of soil contamination at a hazardous waste site should begin with recognition of any qualitative similarities or discernible trends that might be corrected with stratigraphy. Initial perceptions should be validated by actual field sampling.

The ground water investigation, including water quality and hydrological assessment, should occur in coordination with the subsurface soils investigation.

The hydrological portion of the investigation should start with a description of regional and site-specific ground water hydrology. This will include the identification of recharge and discharge areas and rates, presentation of regional and site-specific potentiometric surface contours, estimates of aquifer properties and parameters, and description of the hydrological relationship between zones of concern.

The investigation of water quality condition should include identification and description of plumes of contamination, extent of contamination with reference to known sources of contaminants and direction of ground water flow, vertical stratification of contamination, water quality of upgradient wells, estimates of the rate of movement of contaminants, and potential for contamination of downgradient wells.

Ground water systems are most often complicated and heterogeneous in nature. Distribution and transport analyses should, therefore, be done with an understanding of the inherent limitation and approximations.

Environmental Fate - Biota

The process leading to an accumulation of chemical residues in the body of an organism, above the levels in the environment or food, is termed bioconcentration. Accumulation of chemical residues can occur by direct adsorption from water or air as well as by ingestion. Bioconcentration of chemicals by nonhuman organisms (aquatic or terrestrial plants and animals) is of interest because of the potential for human exposure through consumption of these organisms and the potential direct impact of the chemicals on the accumulating organism. Bioconcentration is a public health concern with food items that may have potentially harmful chemical residues, and thereby pose a risk to the consumer. Methods for estimating or measuring bioconcentration are presented in the Decision Tree Manual.

Environmental Fate - Air

There is increasing evidence that air emissions of toxic chemicals from hazardous waste sites may pose a health threat to persons who live, play or work in the vicinity of the site. Volatile chemicals may be released as gases from a variety of sources including landfills, surface impoundments (also called ponds or lagoons), contaminated land or surface waters, land treatment areas, deteriorating containers or tanks. Chemicals adsorbed to soil may also be transported as windblown particulate matter, especially in areas with frequent vehicular or mechanical disturbance.

The Decision Tree Process provides an analytic framework for assessing the magnitude of existing or potential air contamination which may arise from hazardous waste sites. A complete investigation involves preliminary screening based on chemical and site characterstics followed by the use of calculations and field monitoring approaches to estimate emission rates and air concentrations of toxic chemicals.

The most important chemical parameters to consider when evaluating air emissions from a hazardous waste site are the vapor pressure and the Henry's Law

Constant. Vapor pressure, defined as the pressure exerted by a gas when in equilibrium with the liquid or solid phase, is a useful screening indicator of the potential of a chemical to volatilize from land. The Henry's Law Constant, which describes equilibrium partitioning of a chemical between solution in water and the gas phase of the chemical, is the most relevant parameter to estimate the tendency of a chemical to volatilize from a surface impoundment or water.

For wastes which have been deposited in landfills, mixed in the ground, or have seeped downward from soil surface contamination, Henry's Law Constant, indicates the tendency of the chemical to partition between soil water in the vadose zone or ground water and the soil vapor phase. This partitioning is the first step for volatile air emissions from hazardous wastes beneath the soil surface. The vapor pressure and Henry's Law Constant, however, are not sufficient to provide a good indication of the magnitude of an air emission problem from volatile chemicals. Site-specific characteristics must be considered.

The environmental characterstics of the site are a major factor influencing the potential for, and extent of, an air emission problem. Soil characteristics such as porosity, moisture, and organic content are particularly significant when evaluating volatile emissions from land. Adsorption of a chemical to the soil reduces the extent of volatilization. Precipitation and downward movement also decrese the concentration of the chemical which will reach the air. Meteorologic conditions such as temperature, wind speed, and barometric pressure may influence emission rate from waste sites; other meteorologic characteristics such as wind speed and direction influence the movement of the chemical once it is released and, ultimately, the concentration at biological receptors.

Analytic techniques selected for inclusion in this manual were based upon the accuracy of description of the phenomena and availability of input data. The selected emission rate estimation methods for various types of hazardous waste sites include most of the methods selected by the Environmental Protection Agency. These approaches generally provide conservative estimates of downwind conditions that would not be expected to be exceeded.

Risk Determination

The appraisal of the adverse impacts of toxic substances on biological receptors at concentrations predicted to occur in the future is essentially identical to that employed to evaluate the adverse impacts of existing concentrations of toxic substances. Once a predicted level of contamination is determined, the AALs are employed to appraise the risks associated with the predicted levels of exposure. Should a significant risk be identified, a risk management process should be initiated.

COMPONENT V: Development of Mitigation Strategies and Remedial Action Selection

The Decision Tree Process comes to conclusion in this fifth component. Based on the degree of hazard and the characterstics of the site, alternatives for

remedial action can be identified. It is anticipated that the alternatives will be developed either by the responsible party or by regional contractors working for the State. When appropriate, State staff will develop a preferred alternative.

The objective of site mitigation is to assure that the biological receptors associated with each environmental pathway are not exposed to hazardous chemcials at levels above the Applied Action Levels (AALs). The strategies developed to achieve this objective may include control of the pathway (such as ground water extraction and treatment), modification of the pathway (such as capping a site to reduce infiltration), or control of the source material (such as on-site stabilization or treatment of contaminated soils). The physical, legal, and administrative actions necessary to implement site mitigation and maintain the desired effects of the site mitigation strategy are developed in the Remedial Action Plan (RAP). State staff and regional contractors will evaluate likely remedial alternatives.

Selection of the preferred remedial action should be made based on the scientific and technical evaluations cited in the preceding text. However, local, political, social, and other considerations must also be factored into the final decision. For instance, if a small site of marginal threat to health and environment exists within a widely contaminated industrial setting, the decision makers must consider if the public's best interests are being served through the implementation of an extensive remedial action. Factors that need to be addressed include the availability or unavailability of resources to mitigate other sources of contamination that are of comparable or greater significance, costs of materials and required manpower, the costs of transportation and disposal if soil or water removal is an option, and exposures likely to occur resulting from uncovering buried wastes.

The utilization of the Decision Tree Process in development of site mitigation strategies and RAPs will include a re-evaluation of the site to determine if post-mitigation exposure to hazardous chemicals associated with a particular site will exceed AALs.

CONCLUSION

The California Site Mitigation Decision Tree provides a systematic method for identifying and evaluating the risk associated with abandoned or uncontrolled hazardous waste sites. Because of the rapidly changing nature of scientific and technical knowledge in this area, the Decision Tree process has been designed for flexibility and expandability.

The Decision Tree process provides State decision makers with a logical, systematic, and time efficient approach to mitigating contaminated sites. This represents significant progress in meeting the challenge of protecting the public health and the environment from adverse effects of exposure to toxic chemicals found on these contaminated sites.

I. PRELIMINARY SITE APPRAISAL

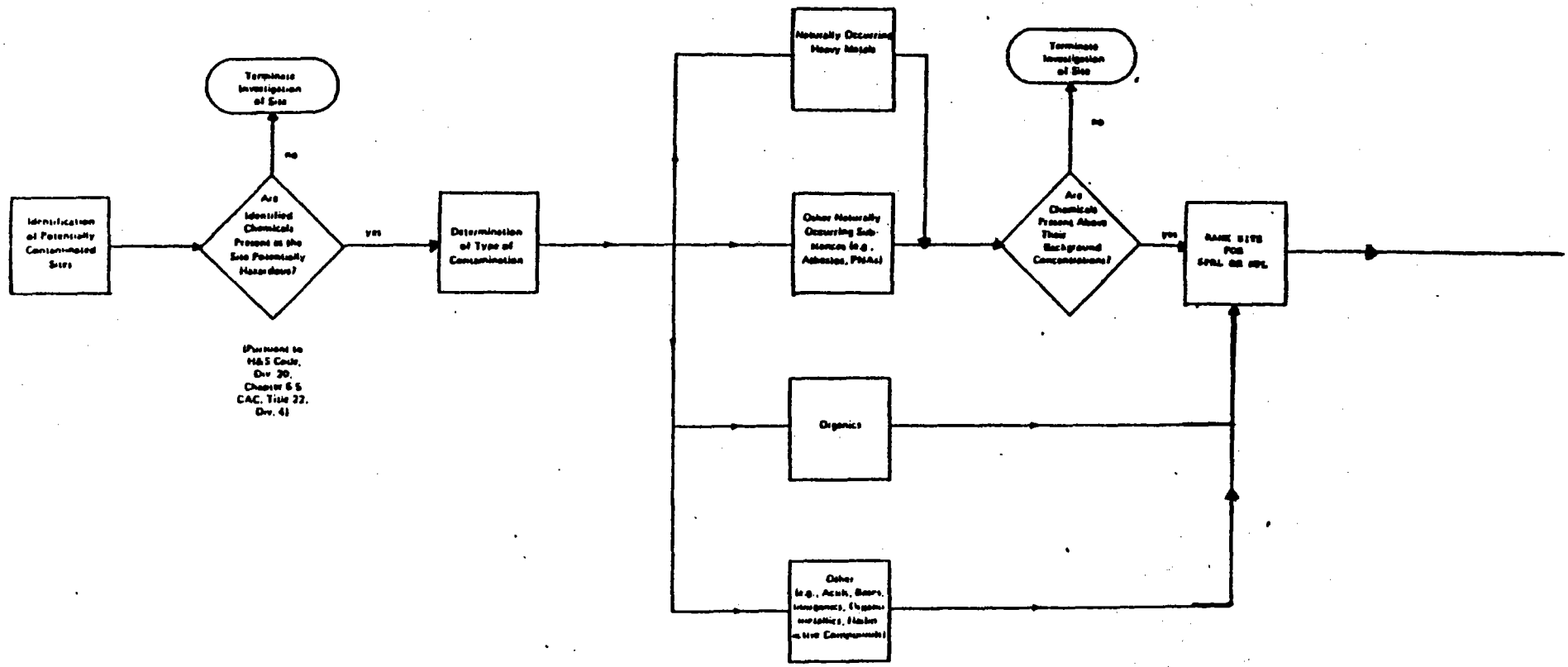
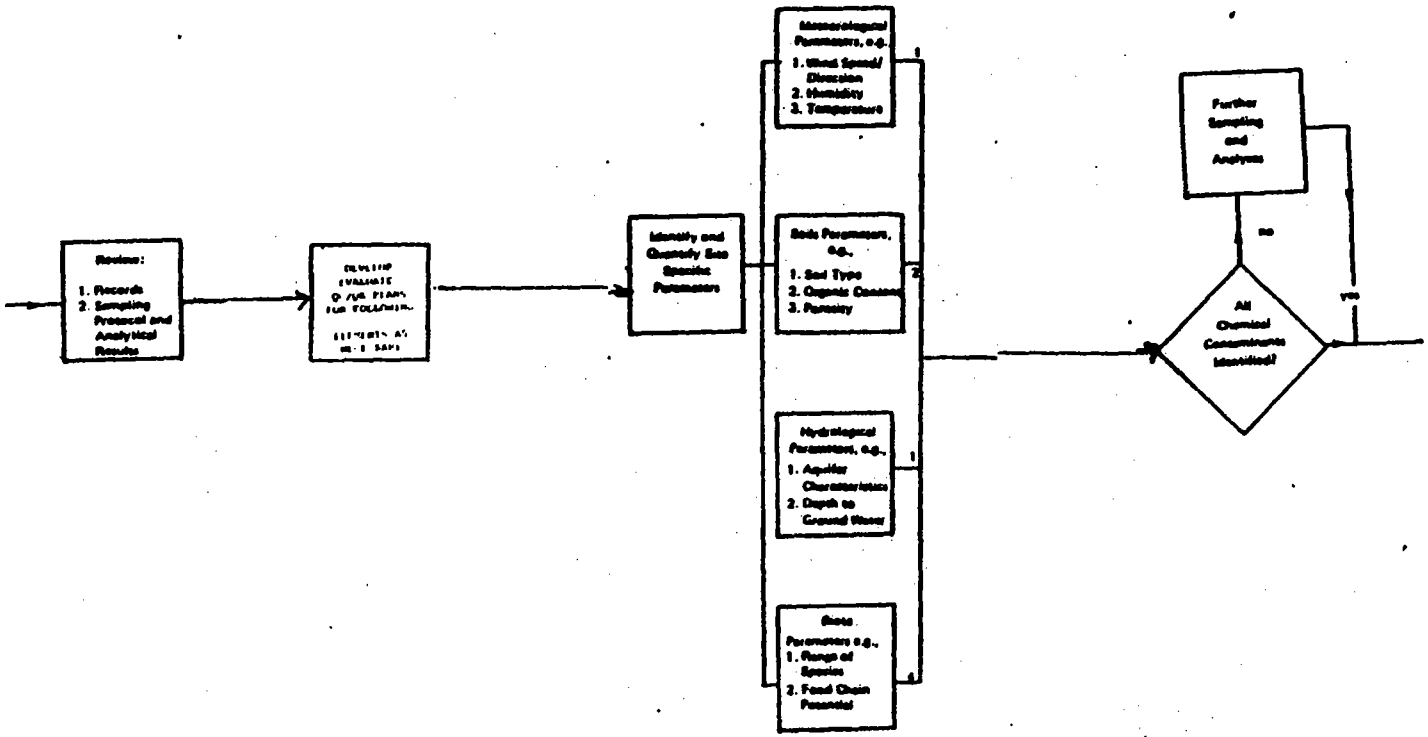
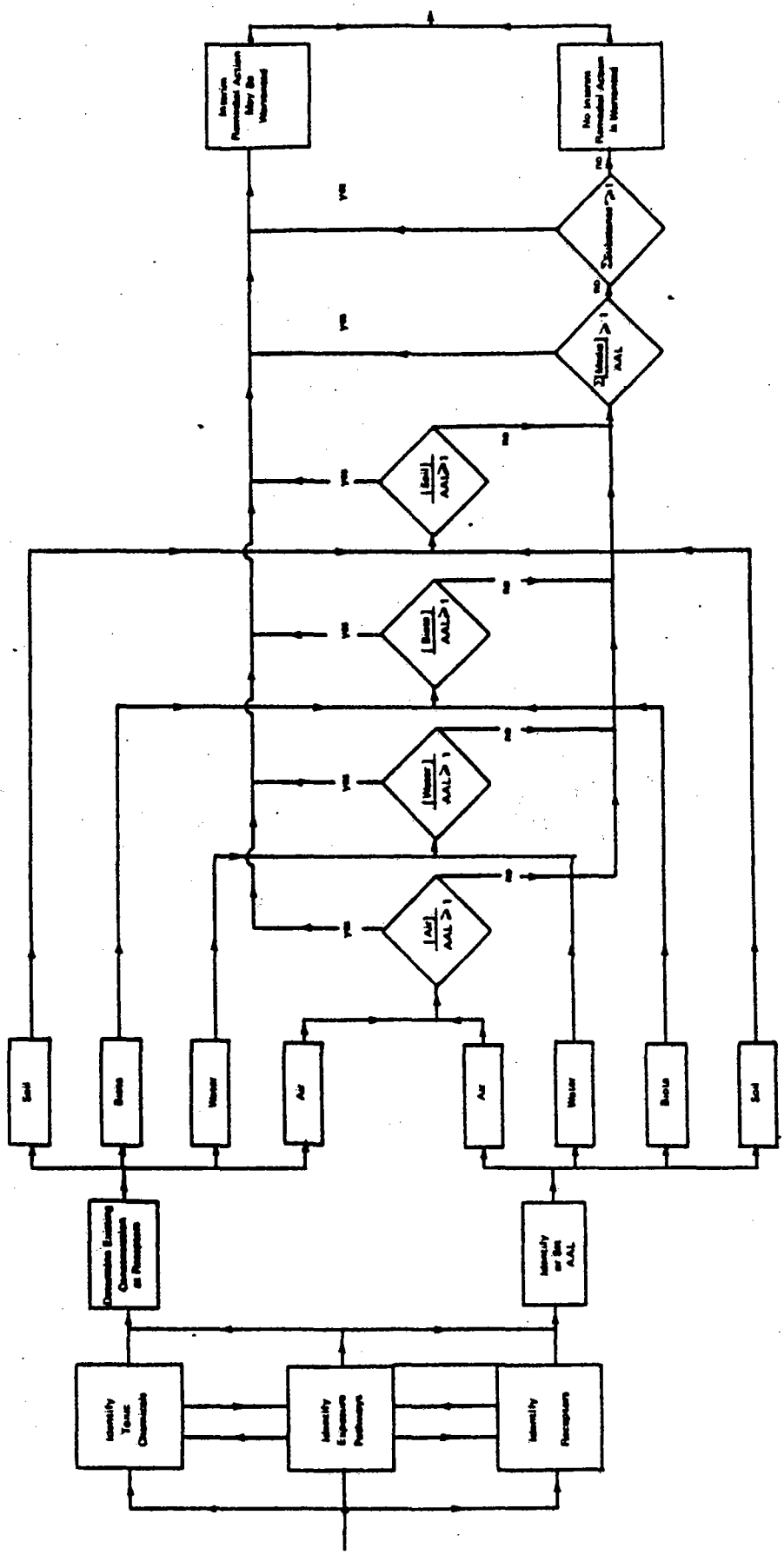
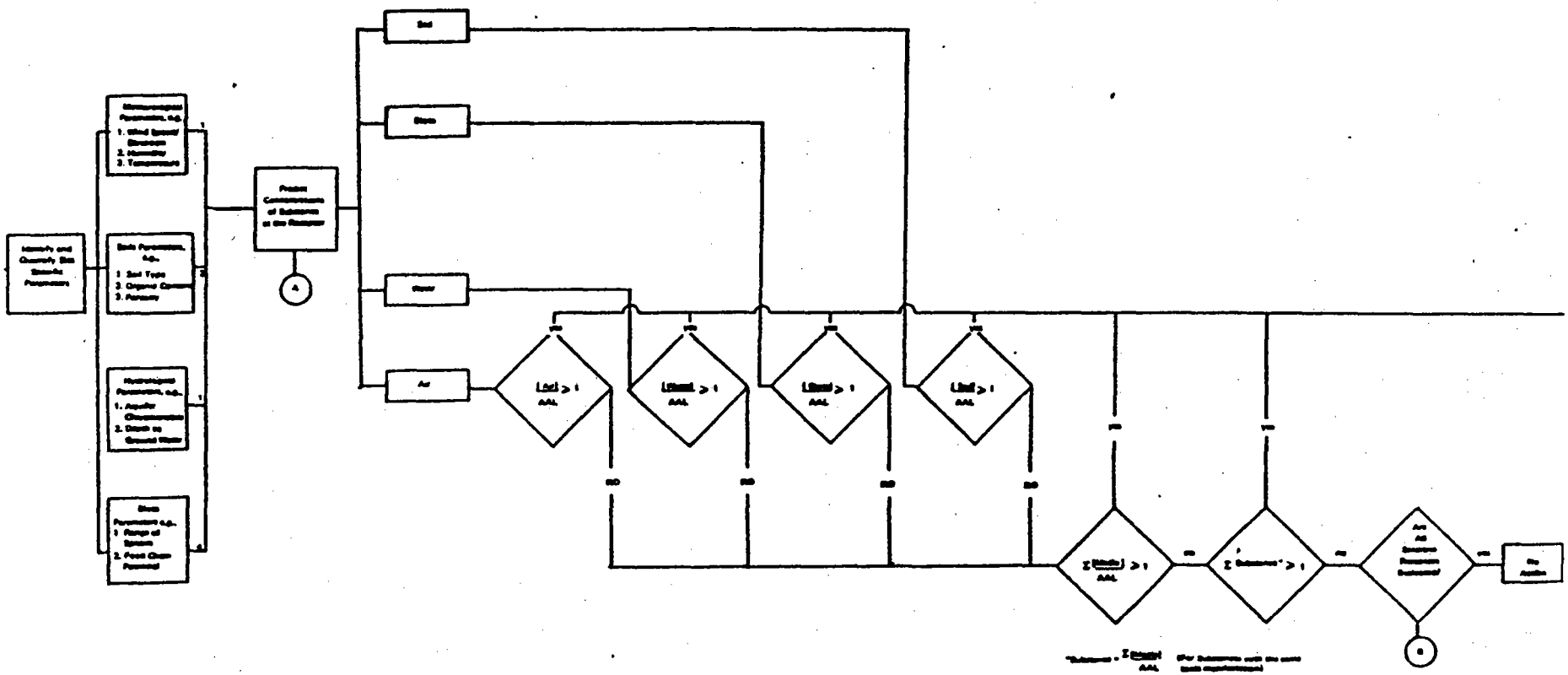


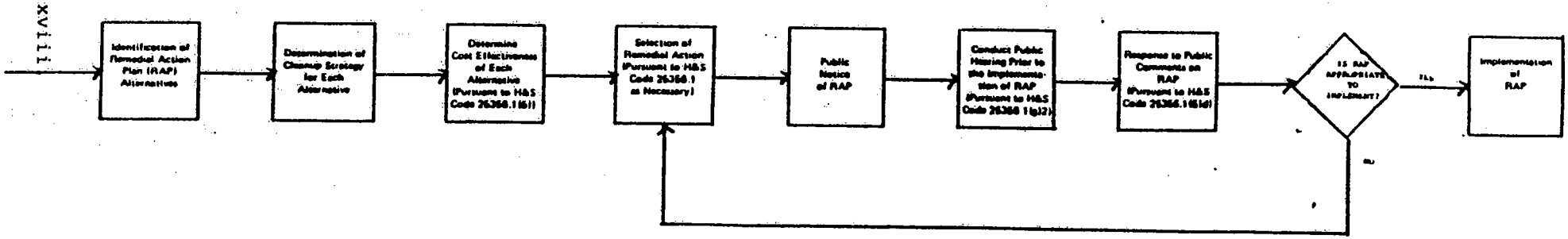
FIGURE 1. OVERVIEW OF THE DECISION TREE


Review:
1. Records
2. Sampling Protocol and Analytical Results
Identify and Quantify Site Specific Parameters
Meteorological Parameters, e.g.
1. Wind Speed/ Direction
2. Humidity
3. Temperature
Soils Parameters, e.g.,
1. Soil Type
2. Organic Content
3. Porosity
Hydrological Parameters, e.g.,
1. Aquifer Characteristics
2. Depth to Ground Water
Biota Parameters e.g.,
1. Ranges of Species
2. Food Chain Potential
Further Sampling and Analysis
No
Yes
All Chemical Contaminants Identified?


Identify Toxic Chemicals
Identify Exposure Pathways
Identify Receptors
Soil
Biota
Water
Air
Air
Water
Biota
Soil



IV. ENVIRONMENTAL FATE AND RISK DETERMINATION



V. DEVELOPMENT OF MITIGATION STRATEGY AND SELECTION OF REMEDIAL ACTI

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CENTRAL COAST REGION (3)
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